



Budd-Chiari syndrome



Budd-Chiari syndrome, or hepatic vein thrombosis, is usually seen in the context of underlying haematological disease or another procoagulant condition

Causes

- polycythaemia rubra vera
- thrombophilia: activated protein C resistance, antithrombin III deficiency, protein C & S deficiencies
- pregnancy
- oral contraceptive pill

Features

- abdominal pain: sudden onset, severe
- ascites
- tender hepatomegaly

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Abdominal pain, ascites, tender hepatomegaly - Budd-Chiari syndrome

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Wilson's disease



Wilson's disease is an autosomal recessive disorder characterised by excessive copper deposition in the tissues. Metabolic abnormalities include increased copper absorption from the small intestine and decreased hepatic copper excretion. Wilson's disease is caused by a defect in the ATP7B gene located on chromosome 13.

The onset of symptoms is usually between 10 - 25 years. Children usually present with liver disease whereas the first sign of disease in young adults is often neurological disease

Features result from excessive copper deposition in the tissues, especially the brain, liver and cornea:

- liver: hepatitis, cirrhosis
- neurological: basal ganglia degeneration, speech and behavioural problems are often the first manifestations. Also: asterixis, chorea, dementia
- Kayser-Fleischer rings
- renal tubular acidosis (esp. Fanconi syndrome)
- haemolysis
- blue nails

Diagnosis

- reduced serum caeruloplasmin
- reduced serum copper (counter-intuitive, but 95% of plasma copper is carried by caeruloplasmin)
- increased 24hr urinary copper excretion

Management

- penicillamine (chelates copper) has been the traditional first-line treatment
- trientine hydrochloride is an alternative chelating agent which may become first-line treatment in the future
- tetrathiomolybdate is a newer agent that is currently under investigation

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Less relevant

Alpha-1 antitrypsin deficiency

Alpha-1 antitrypsin (A1AT) deficiency is a common inherited condition caused by a lack of a protease inhibitor (Pi) normally produced by the liver. The role of A1AT is to protect cells from enzymes such as neutrophil elastase.

Genetics

- located on chromosome 14
- inherited in an autosomal recessive / co-dominant fashion*
- alleles classified by their electrophoretic mobility - M for normal, S for slow, and Z for very slow
- normal = PiMM
- homozygous PiSS (50% normal A1AT levels)
- homozygous PiZZ (10% normal A1AT levels)

Features

- patients who manifest disease usually have PiZZ genotype
- lungs: panacinar emphysema, most marked in lower lobes
- liver: cirrhosis and hepatocellular carcinoma in adults, cholestasis in children

Investigations

- A1AT concentrations

Management

- no smoking
- supportive: bronchodilators, physiotherapy
- intravenous alpha1-antitrypsin protein concentrates
- surgery: volume reduction surgery, lung transplantation

*trusted sources are split on which is a more accurate description

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Haemochromatosis: features

Haemochromatosis is an autosomal recessive disorder of iron absorption and metabolism resulting in iron accumulation. It is caused by inheritance of mutations in the HFE gene on both copies of chromosome 6*. It is often asymptomatic in early disease and initial symptoms often non-specific e.g. lethargy and arthralgia

Epidemiology

- 1 in 10 people of European descent carry a mutation genes affecting iron metabolism, mainly HFE
- prevalence in people of European descent = 1 in 200

Presenting features

- early symptoms include fatigue, erectile dysfunction and arthralgia (often of the hands)
- 'bronze' skin pigmentation
- diabetes mellitus
- liver: stigmata of chronic liver disease, hepatomegaly, cirrhosis, hepatocellular deposition)
- cardiac failure (2nd to dilated cardiomyopathy)
- hypogonadism (2nd to cirrhosis and pituitary dysfunction - hypogonadotrophic hypogonadism)
- arthritis (especially of the hands)

Questions have previously been asked regarding which features are reversible with treatment:

Reversible complications	Irreversible complications
• Cardiomyopathy • Skin pigmentation	• Liver cirrhosis** • Diabetes mellitus • Hypogonadotrophic hypogonadism • Arthropathy

*there are rare cases of families with classic features of genetic haemochromatosis but no mutation in the HFE gene

**whilst elevated liver function tests and hepatomegaly may be reversible, cirrhosis is not

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Haemochromatosis: investigation

Haemochromatosis is an autosomal recessive disorder of iron absorption and metabolism resulting in iron accumulation. It is caused by inheritance of mutations in the HFE gene on both copies of chromosome 6*.

There is continued debate about the best investigation to screen for haemochromatosis.

- general population: transferrin saturation is considered the most useful marker. Ferritin should also be measured but is not usually abnormal in the early stages of iron accumulation
- testing family members: genetic testing for HFE mutation

These guidelines may change as HFE gene analysis become less expensive

Diagnostic tests

- molecular genetic testing for the C282Y and H63D mutations
- liver biopsy: Perl's stain

Typical iron study profile in patient with haemochromatosis

- transferrin saturation > 55% in men or > 50% in women
- raised ferritin (e.g. > 500 ug/l) and iron
- low TIBC

Monitoring adequacy of venesection

- transferrin saturation should be kept below 50% and the serum ferritin concentration below 50 ug/l

Joint x-rays characteristically show chondrocalcinosis

*there are rare cases of families with classic features of genetic haemochromatosis but no mutation in the HFE gene

Next question >

Ferritin

Ferritin is an intracellular protein that binds iron and stores it to be released in a controlled fashion at sites where iron is required.

Increased ferritin levels

This is typically defined as $> 300 \mu\text{g/L}$ in men/postmenopausal women and $> 200 \mu\text{g/L}$ in premenopausal women.

Ferritin is an acute phase protein and may be synthesised in increased quantities in situations where inflammatory activity is ongoing. Falsely elevated results may therefore be encountered clinically and need to be taken in the context of the clinical picture and blood results.

We can split the causes of increased ferritin levels into 2 distinct categories;

Without iron overload (around 90% of patients)	With iron overload (around 10% of patients)
Inflammation (due to ferritin being an acute phase reactant) Alcohol excess Liver disease Chronic kidney disease Malignancy	Primary iron overload (hereditary haemochromatosis) Secondary iron overload (e.g. following repeated transfusions)

The best test to see whether iron overload is present is **transferrin saturation**. Typically, normal values of $< 45\%$ in females and $< 50\%$ in males exclude iron overload.

Reduced ferritin levels

Because iron and ferritin are bound the total body ferritin levels may be decreased in cases of iron deficiency anaemia.

Measurement of serum ferritin levels can be useful in determining whether an apparently low haemoglobin and microcytosis is truly caused by an iron deficiency state.



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Spontaneous bacterial peritonitis

Spontaneous bacterial peritonitis (SBP) is a form of peritonitis usually seen in patients with ascites secondary to liver cirrhosis.

Diagnosis

- paracentesis: neutrophil count > 250 cells/ul

Management

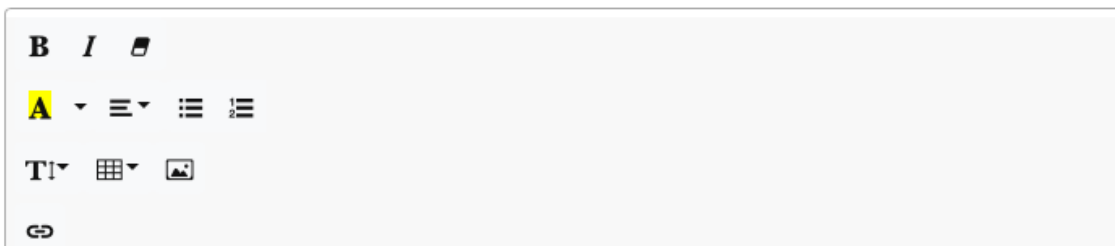
- intravenous cefotaxime is usually given

Antibiotic prophylaxis should be given to patients with ascites if:

- patients who have had an episode of SBP
- patients with fluid protein <15 g/l and either Child-Pugh score of at least 9 or hepatorenal syndrome
- NICE recommend: 'Offer prophylactic oral ciprofloxacin or norfloxacin for people with cirrhosis and ascites with an ascitic protein of 15 g/litre or less, until the ascites has resolved'

Alcoholic liver disease is a marker of poor prognosis in SBP.

Next question >



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Questions Lab Values, Normal Adult: Laboratory Reference Ranges in H...

gastrointestinal signs. Proton-pump inhibitors like Lansoprazole do play a role, if the patient is complaining of dyspeptic like symptoms. Antibiotics and pancreatic enzyme replacements play no part in management.



 Discuss
  Improve

Next question >

Post-cholecystectomy syndrome

Post-cholecystectomy syndrome is a recognised complication of cholecystectomies. Typically symptoms of dyspepsia, vomiting, pain, flatulence and diarrhoea occur in up to 40% patients post surgery.

The pathology behind the syndrome isn't completely clear, however there is some association with remnant stones and biliary injury. Pain is often due to sphincter of Oddi dysfunction and the development of surgical adhesions.

Management is often difficult, but often involves a low-fat diet and the introduction of bile acid sequestrants, such as Cholestyramine, to bind the excess bile acids and thus preventing lower gastrointestinal signs. Proton-pump inhibitors like Lansoprazole do play a role, if the patient is complaining of dyspeptic like symptoms. Antibiotics and pancreatic enzyme replacements play no part in management.

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Pass Gallstones



Question

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Up to 24% of women and 12% of men may have gallstones. Of these up to 30% may develop local infection and cholecystitis. In patients subjected to surgery, 12% will have stones contained within the common bile duct. The majority of gallstones are of a mixed composition (50%) with pure cholesterol stones accounting for 20% of cases.

The aetiology of CBD stones differs in the world, in the West most CBD stones are the result of migration. In the East, a far higher proportion arise in the CBD de novo.

The classical symptoms are of colicky right upper quadrant pain that occurs postprandially. The symptoms are usually worst following a fatty meal when cholecystokinin levels are highest and gallbladder contraction is maximal.

Investigation

In almost all suspected cases the standard diagnostic workup consists of abdominal ultrasound and liver function tests. Of patients who have stones within the bile duct, 60% will have at least one abnormal result on LFT's. Ultrasound is an important test, but is operator dependent and therefore may occasionally need to be repeated if a negative result is at odds with the clinical picture. Where stones are suspected in the bile duct the options lie between magnetic resonance cholangiography and intraoperative imaging. The choice between these two options is determined by the skills and experience of the surgeon. The advantages of intraoperative imaging are less useful in making therapeutic decisions if the operator is unhappy about proceeding the bile duct exploration, and in such circumstances, preoperative MRCP is probably a better option.

Specific gallstone and gallbladder related disease

Disease	Features	Management
Biliary colic	Colicky abdominal pain, worse postprandially, worse after fatty foods	If imaging shows gallstones and history compatible then laparoscopic cholecystectomy
Acute cholecystitis	Right upper quadrant pain Fever Murphys sign on examination Occasionally mildlv	Imaging (USS) and cholecystectomy (ideally within 48 hours of presentation) (2)



Pass	Acute cholecystitis	Right upper quadrant pain Fever Murphys sign on examination Occasionally mildly deranged LFT's (especially if Mirizzi syndrome)	Imaging (USS) and cholecystectomy (ideally within 48 hours of presentation) (2)	session
Question	Gallbladder abscess	Usually prodromal illness and right upper quadrant pain Swinging pyrexia Patient may be systemically unwell Generalised peritonism not present	Imaging with USS +/- CT Scanning Ideally, surgery although subtotal cholecystectomy may be needed if Calot's triangle is hostile In unfit patients, percutaneous drainage may be considered	
What is the	Cholangitis	Patient severely septic and unwell Jaundice Right upper quadrant pain	Fluid resuscitation Broad-spectrum intravenous antibiotics Correct any coagulopathy Early ERCP	
Dix-Halliday	Gallstone ileus	Patients may have a history of previous cholecystitis and known gallstones Small bowel obstruction (may be intermittent)	Laparotomy and removal of the gallstone from small bowel, the enterotomy must be made proximal to the site of obstruction and not at the site of obstruction. The fistula between the gallbladder and duodenum should not be interfered with.	(1/1)
Metronidazole	Acalculous	Patients with	If patient fit then cholecystectomy, if unfit then	(1/1)
Senna				(1/1)
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Propranolol				(1/1)
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Acalculous
cholecystitis

Patients with
intercurrent
illness (e.g.
diabetes,
organ failure)
Patient of
systemically
unwell
Gallbladder
inflammation in
absence of
stones
High fever

If patient fit then cholecystectomy, if unfit then
percutaneous cholecystostomy

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Treatment

Asymptomatic gallstones which are located in the gallbladder are common and do not require treatment. However, if stones are present in the common bile duct there is an increased risk of complications such as cholangitis or pancreatitis and surgical management should be considered.

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Female

Choles

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Patients with asymptomatic gallstones rarely develop symptoms related to them (less than 2% per year) and may, therefore, be managed expectantly. In almost all cases of symptomatic gallstones the treatment of choice is cholecystectomy performed via the laparoscopic route. In the very frail patient, there is sometimes a role for the selective use of ultrasound guided cholecystostomy.

During the course of the procedure, some surgeons will routinely perform either intraoperative cholangiography to either confirm anatomy or to exclude CBD stones. The latter may be more easily achieved by use of laparoscopic ultrasound. If stones are found then the options lie between early ERCP in the day or so following surgery or immediate surgical exploration of the bile duct. When performed via the trans cystic route this adds little in the way of morbidity and certainly results in faster recovery. Where transcystic exploration fails the alternative strategy is that of formal choledochotomy. The exploration of a small duct is challenging and ducts of less than 8mm should not be explored. Small stones that measure less than 5mm may be safely left and most will pass spontaneously.

Risks of ERCP(1)

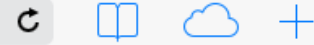
- Bleeding 0.9% (rises to 1.5% if sphincterotomy performed)
- Duodenal perforation 0.4%
- Cholangitis 1.1%
- Pancreatitis 1.5%

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- Cholangitis 1.1%
- Pancreatitis 1.5%

References

1. Williams E *et al.* Guidelines on the management of common bile duct stones (CBDS)*Gut* 2008;57:10041021
2. Gurusamy KS, Samraj K. Early versus delayed laparoscopic cholecystectomy for acute cholecystitis. *Cochrane Database Syst Rev.* 2006 Oct 18;(4):CD005440.



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[2014 Gallstone disease: diagnosis and management](#)[Biliary colic](#)

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Questions

Lab Values, Normal Adult: Laboratory Reference Ranges in H...

The dry mouth is this patient is due to sicca syndrome.

Primary biliary cirrhosis. The raised alkaline phosphatase point towards a diagnosis primary biliary cirrhosis rather than primary Sjogren's syndrome.

Discuss (1) Improve

Next question >

Primary biliary cirrhosis: features

Primary biliary cirrhosis (now increasingly referred to as primary biliary cholangitis) is a chronic liver disorder typically seen in middle-aged females (female:male ratio of 9:1). The aetiology is not fully understood although it is thought to be an autoimmune condition. Interlobular bile ducts become damaged by a chronic inflammatory process causing progressive cholestasis, which may eventually progress to cirrhosis. The classic presentation is itching in a middle-aged woman

Clinical features

- early: may be asymptomatic (e.g. raised ALP on routine LFTs) or fatigue, pruritus
- cholestatic jaundice
- hyperpigmentation, especially over pressure points
- xanthelasmas, xanthomata
- also: clubbing, hepatosplenomegaly
- late: may progress to liver failure

Complications

- malabsorption: osteomalacia, coagulopathy
- sicca syndrome occurs in 70% of cases
- portal hypertension: ascites, variceal haemorrhage
- hepatocellular cancer (20-fold increased risk)

Next question >

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Less relevant

Primary sclerosing cholangitis

Primary sclerosing cholangitis is a biliary disease of unknown aetiology characterised by inflammation and fibrosis of intra and extra-hepatic bile ducts

Associations

- ulcerative colitis: 4% of patients with UC have PSC, 80% of patients with PSC have UC
- Crohn's (much less common association than UC)
- HIV

Features

- cholestasis: jaundice and pruritus
- right upper quadrant pain
- fatigue

Investigation

- ERCP is the standard diagnostic tool, showing multiple biliary strictures giving a 'beaded' appearance
- ANCA may be positive
- there is a limited role for liver biopsy, which may show fibrous, obliterative cholangitis often described as 'onion skin'

Complications

- cholangiocarcinoma (in 10%)
- increased risk of colorectal cancer

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Relevant to my revision >

Less relevant >

Ascending cholangitis

Ascending cholangitis is a bacterial infection of the biliary tree. The most common predisposing factor is gallstones.

Charcot's triad of right upper quadrant (RUQ) pain, fever and jaundice occurs in about 20-50% of patients

- fever is the most common feature, seen in 90% of patients
- RUQ pain 70%
- jaundice 60%
- hypotension and confusion are also common

Management

- intravenous antibiotics
- endoscopic retrograde cholangiopancreatography (ERCP) after 24-48 hours to relieve any obstruction

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Inherited causes of jaundice

There are 4 inherited causes of jaundice you need to be aware of: Gilbert's syndrome, Crigler-Najjar syndrome, Dubin-Johnson syndrome and Rotor's syndrome. It is important for the exam to be able to classify them according to whether they cause conjugated or unconjugated hyperbilirubinaemia:

Unconjugated hyperbilirubinaemia	Conjugated hyperbilirubinaemia
Gilbert's syndrome Crigler-Najjar syndrome	Dubin-Johnson syndrome Rotor syndrome

Important points for the exam:

Gilbert's syndrome

- autosomal recessive
- UDP-glucuronyl transferase deficiency causing defective conjugation
- benign

Crigler-Najjar syndrome, type 1

- autosomal recessive
- absolute deficiency of UDP-glucuronosyl transferase
- do not survive to adulthood

Crigler-Najjar syndrome, type 2

- slightly more common than type 1 and less severe
- may improve with phenobarbital

Dubin-Johnson syndrome

- autosomal recessive. Relatively common in Iranian Jews
- mutation in the canalicular multidrug resistance protein 2 (MRP2) results in defective hepatic excretion of bilirubin
- results in a grossly black liver
- benign

Rotor syndrome

- autosomal recessive
- defect in the hepatic uptake and storage of bilirubin
- benign

Question 14

Right up

Gilbert

Biliary

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Isolated

Coeliac disease - anti-gliadin antibody

Dysphagia (oesophageal webs), glossitis, iron-deficiency anaemia - Plummer-Vinson syndrome

Multiple colonic polyps, osteomas, epidermoid cysts - Gardner's syndrome

Gilbert's syndrome



Gilbert's syndrome is an autosomal recessive* condition of defective bilirubin conjugation due to a deficiency of UDP glucuronyl transferase. The prevalence is approximately 1-2% in the general population

Features

- unconjugated hyperbilinaemia (i.e. not in urine)
- jaundice may only be seen during an intercurrent illness

Investigation and management

- investigation: rise in bilirubin following prolonged fasting or IV nicotinic acid
- no treatment required

*the exact mode of inheritance is still a matter of debate

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Hepatorenal syndrome: management

The management of hepatorenal syndrome (HRS) is notoriously difficult. The ideal treatment is liver transplantation but patients are often too unwell to have surgery and there is a shortage of donors

The most accepted theory regarding the pathophysiology of HRS is that vasoactive mediators cause **splanchnic vasodilation** which in turn reduces the systemic vascular resistance. This results in 'underfilling' of the kidneys. This is sensed by the juxtaglomerular apparatus which then activates the renin-angiotensin-aldosterone system, causing renal vasoconstriction which is not enough to counterbalance the effects of the splanchnic vasodilation.

Hepatorenal syndrome has been categorized into two types:

Type 1 HRS	Type 2 HRS
Rapidly progressive Doubling of serum creatinine to > 221 $\mu\text{mol/L}$ or a halving of the creatinine clearance to less than 20 ml/min over a period of less than 2 weeks Very poor prognosis	Slowly progressive Prognosis poor, but patients may live for longer

Management options

- vasopressin analogues, for example terlipressin, have a growing evidence base supporting their use. They work by causing vasoconstriction of the splanchnic circulation
- volume expansion with 20% albumin
- transjugular intrahepatic portosystemic shunt

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Less relevant >

Bile-acid malabsorption

Bile-acid malabsorption is a cause of chronic diarrhoea. This may be primary, due to an excessive production of bile acid, or secondary to an underlying gastrointestinal disorder causing reduced bile acid absorption. Secondary causes are often seen in patients with ileal disease, such as with Crohn's.

Other secondary causes include:

- cholecystectomy
- coeliac disease
- small intestinal bacterial overgrowth

Investigation

- the test of choice is **SeHCAT**
- nuclear medicine test using a gamma-emitting selenium molecule in selenium homocholic acid taurine or tauroselcholic acid (SeHCAT)
- scans are done 7 days apart to assess the retention/loss of radiolabelled $^{75}\text{SeHCAT}$

Management

- bile acid sequestrants e.g. cholestyramine

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Relevant to my revision

Less relevant

Hepatitis B serology

Interpreting hepatitis B serology is a dying art form which still occurs at regular intervals in medical exams. It is important to remember a few key facts:

- surface antigen (HBsAg) is the first marker to appear and causes the production of anti-HBs
- HBsAg normally implies acute disease (present for 1-6 months)
- if HBsAg is present for > 6 months then this implies chronic disease (i.e. Infective)
- Anti-HBs implies immunity (either exposure or immunisation). It is negative in chronic disease
- Anti-HBc implies previous (or current) infection. IgM anti-HBc appears during acute or recent hepatitis B infection and is present for about 6 months. IgG anti-HBc persists
- HbeAg results from breakdown of core antigen from infected liver cells as is therefore a marker of infectivity

Example results

- previous immunisation: anti-HBs positive, all others negative
- previous hepatitis B (> 6 months ago), not a carrier: anti-HBc positive, HBsAg negative
- previous hepatitis B, now a carrier: anti-HBc positive, HBsAg positive

HBsAg = ongoing infection, either acute or chronic if present > 6 months

anti-HBc = caught, i.e. negative if immunized

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Questions	Lab Values, Normal Adult: Laboratory Reference Ranges in H...
Without intervention the vertical transmission rate is around 3%	14%
Only at risk groups should be screened for hepatitis B during pregnancy	9%
Around 30% of mothers with hepatitis B develop pre-eclampsia	5%
It is safe for a mother with hepatitis B to breastfeed her newborn	55%
All pregnant women with hepatitis B should take oral ribavirin in the last trimester of pregnancy	17%

Without intervention the vertical transmission rate is around 20%, which increases to 90% if the woman is positive for HBeAg.

Discuss

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Hepatitis B and pregnancy

Basics

- all pregnant women are offered screening for hepatitis B
- babies born to mothers who are chronically infected with hepatitis B or to mothers who've had acute hepatitis B during pregnancy should receive a complete course of vaccination + hepatitis B immunoglobulin
- studies are currently evaluating the role of oral antiviral treatment (e.g. Lamivudine) in the latter part of pregnancy
- there is little evidence to suggest caesarean section reduces vertical transmission rates
- hepatitis B cannot be transmitted via breastfeeding (in contrast to HIV)

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Hepatitis D

Hepatitis D is a single stranded RNA virus that is transmitted parenterally. It is an incomplete RNA virus that requires hepatitis B surface antigen to complete its replication and transmission cycle.

It is transmitted in a similar fashion to hepatitis B (exchange of bodily fluids) and patients may be infected with hepatitis B and hepatitis D at the same time.

Hepatitis D terminology:

- Co-infection: Hepatitis B and Hepatitis D infection at the same time.
- Superinfection: A hepatitis B surface antigen positive patient subsequently develops a hepatitis D infection.

Superinfection is associated with high risk of fulminant hepatitis, chronic hepatitis status and cirrhosis.

Diagnosis is made via reverse polymerase chain reaction of hepatitis D RNA. Interferon is currently used as treatment, but with a poor evidence base.

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29 facts found

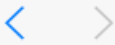
Hepatobiliary disease and related disorders

The table below gives characteristic exam question features for conditions causing hepatobiliary disease and related disorders:

Condition	Features
Viral hepatitis	Common symptoms include: • nausea and vomiting, anorexia • myalgia • lethargy • right upper quadrant (RUQ) pain Questions may point to risk factors such as foreign travel or intravenous drug use.
Congestive hepatomegaly	The liver only usually causes pain if stretched. One common way this can occur is as a consequence of congestive heart failure. In severe cases cirrhosis may occur.
Biliary colic	RUQ pain, intermittent, usually begins abruptly and subsides gradually. Attacks often occur after eating. Nausea is common. It is sometimes taught that patients are female, forties, fat and fair although this is obviously a generalisation.
Acute cholecystitis	Pain similar to biliary colic but more severe and persistent. The pain may radiate to the back or right shoulder. The patient may be pyrexial and Murphy's sign positive (arrest of inspiration on palpation of the RUQ)
Ascending cholangitis	An infection of the bile ducts commonly secondary to gallstones. Classically presents with a triad of: • fever (rigors are common) • RUQ pain • jaundice
Gallstone ileus	This describes small bowel obstruction secondary to an impacted gallstone. It may develop if a fistula forms between a gangrenous gallbladder and the duodenum. Abdominal pain, distension and vomiting are seen.
Cholangiocarcinoma	Persistent biliary colic symptoms, associated with anorexia, jaundice and weight loss. A palpable mass in the right upper quadrant (Courvoisier sign), periumbilical lymphadenopathy (Sister Mary Joseph nodes) and left supraclavicular adenopathy

Gallstone ileus	This describes small bowel obstruction secondary to an impacted gallstone. It may develop if a fistula forms between a gangrenous gallbladder and the duodenum. Abdominal pain, distension and vomiting are seen.
Cholangiocarcinoma	Persistent biliary colic symptoms, associated with anorexia, jaundice and weight loss. A palpable mass in the right upper quadrant (Courvoisier sign), periumbilical lymphadenopathy (Sister Mary Joseph nodes) and left supraclavicular adenopathy (Virchow node) may be seen
Acute pancreatitis	Usually due to alcohol or gallstones Severe epigastric pain Vomiting is common Examination may reveal tenderness, ileus and low-grade fever Periumbilical discolouration (Cullen's sign) and flank discolouration (Grey-Turner's sign) is described but rare
Pancreatic cancer	Painless jaundice is the classical presentation of pancreatic cancer. However pain is actually a relatively common presenting symptom of pancreatic cancer. Anorexia and weight loss are common
Amoebic liver abscess	Typical symptoms are malaise, anorexia and weight loss. The associated RUQ pain tends to be mild and jaundice is uncommon.

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Non-alcoholic fatty liver disease



Non-alcoholic fatty liver disease (NAFLD) is now the most common cause of liver disease in the developed world. It is largely caused by obesity and describes a spectrum of disease ranging from:

- steatosis - fat in the liver
- steatohepatitis - fat with inflammation, non-alcoholic steatohepatitis (NASH), see below
- progressive disease may cause fibrosis and liver cirrhosis

NAFLD is thought to represent the hepatic manifestation of the metabolic syndrome and hence insulin resistance is thought to be the key mechanism leading to steatosis

Non-alcoholic steatohepatitis (NASH) is a term used to describe liver changes similar to those seen in alcoholic hepatitis in the absence of a history of alcohol abuse. It is relatively common and thought to affect around 3-4% of the general population. The progression of disease in patients with NASH may be responsible for a proportion of patients previously labelled as cryptogenic cirrhosis

Associated factors

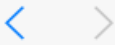
- obesity
- hyperlipidaemia
- type 2 diabetes mellitus
- jejunoileal bypass
- sudden weight loss/starvation

Features

- usually asymptomatic
- hepatomegaly
- ALT is typically greater than AST
- increased echogenicity on ultrasound

NICE produced guidelines on the investigation and management of NAFLD in 2016. Key points:

- there is no evidence to support screening for NAFLD in adults, even in at risk groups (e.g. type 2 diabetes)
- the guidelines are therefore based on the management of the *incidental* finding of NAFLD - typically asymptomatic fatty changes on liver ultrasound
- in these patients, NICE recommends the use of the enhanced liver fibrosis (ELF) blood test to check for advanced fibrosis
- the ELF blood test is a combination of hyaluronic acid + procollagen III + tissue inhibitor of metalloproteinase 1. An algorithm based on these values results in an ELF blood test score, similar to triple testing for Down's syndrome



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NICE produced guidelines on the investigation and management of NAFLD in 2016. Key points:

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Management

- the mainstay of treatment is lifestyle changes (particularly weight loss) and monitoring
- there is ongoing research into the role of gastric banding and insulin-sensitising drugs (e.g. metformin)

B*I*U**A****T!**[Save my notes](#)

NICE

0 1

[2016 Non-alcoholic fatty liver disease \(NAFLD\): assessment and management](#)

[Non-alcoholic fatty liver disease \(NAFLD\) - causes, symptoms, diagnosis, treatment, pathology](#)

Osmosis - YouTube

17 1



Hepatocellular carcinoma



Hepatocellular carcinoma (HCC) is the third most common cause of cancer worldwide. Chronic hepatitis B is the most common cause of HCC worldwide with chronic hepatitis C being the most common cause in Europe.

The main risk factor for developing HCC is liver cirrhosis, for example secondary* to hepatitis B & C, alcohol, haemochromatosis and primary biliary cirrhosis. Other risk factors include:

- alpha-1 antitrypsin deficiency
- hereditary tyrosinosis
- glycogen storage disease
- aflatoxin
- drugs: oral contraceptive pill, anabolic steroids
- porphyria cutanea tarda
- male sex
- diabetes mellitus, metabolic syndrome

Features

- tends to present late
- features of liver cirrhosis or failure may be seen: jaundice, ascites, RUQ pain, hepatomegaly, pruritus, splenomegaly
- possible presentation is decompensation in a patient with chronic liver disease

Screening with ultrasound (+/- alpha-fetoprotein) should be considered for high risk groups such as:

- patients liver cirrhosis secondary to hepatitis B & C or haemochromatosis
- men with liver cirrhosis secondary to alcohol

Management options

- early disease: surgical resection
- liver transplantation
- radiofrequency ablation
- transarterial chemoembolisation
- sorafenib: a multikinase inhibitor

*Wilson's disease is an exception

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Next question >

Coagulopathy of liver disease

In liver failure all clotting factors are low, except for factor VIII which is paradoxically supra-normal. This is because factor VIII is synthesised in endothelial cells throughout the body, unlike the other clotting factors which are synthesised purely in hepatic endothelial cells. Furthermore, whilst activated factor VIII is usually rapidly cleared from the blood stream, good hepatic function is required for this to occur, further leading to increases in circulating factor VIII. This is one of many reasons why, despite conventional coagulation studies (increased PT, APTT, decreased fibrinogen) suggesting increased bleeding risk, patients with chronic liver disease are not protected from venous thrombosis formation but are paradoxically at an increased risk of thrombosis, in addition to bleeding. Other events occurring in chronic liver disease which predispose patients to thrombosis formation include reduced synthesis of the purely hepatic derived natural anticoagulants protein c and protein s (vitamin k dependent), and anti-thrombin (non-vitamin k dependent).

Reference

Tripodi et al. An imbalance of pro- vs anti-coagulation factors in plasma from patients with cirrhosis. Gastroenterology. 2009 Dec;137(6):2105-11.

Next question >

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will lie about their alcohol intake.

The recent holiday in Thailand is a distractor.

  [Discuss \(2\)](#) [Improve](#)

[Next question >](#)

Hepatomegaly

Common causes of hepatomegaly

- Cirrhosis: if early disease, later liver decreases in size. Associated with a non-tender, firm liver
- Malignancy: metastatic spread or primary hepatoma. Associated with a hard, irregular. liver edge
- Right heart failure: firm, smooth, tender liver edge. May be pulsatile

Other causes

- viral hepatitis
- glandular fever
- malaria
- abscess: pyogenic, amoebic
- hydatid disease
- haematological malignancies
- haemochromatosis
- primary biliary cirrhosis
- sarcoidosis, amyloidosis

[Next question >](#)

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Next question >

Acute liver failure

Acute liver failure describes the rapid onset of hepatocellular dysfunction leading to a variety of systemic complications.

Causes

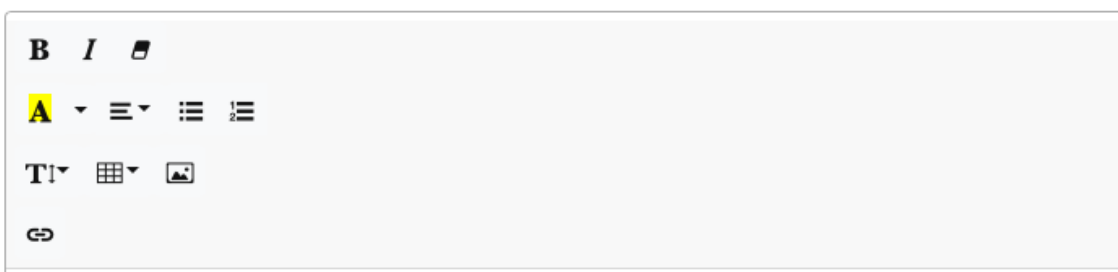
- paracetamol overdose
- alcohol
- viral hepatitis (usually A or B)
- acute fatty liver of pregnancy

Features*

- jaundice
- coagulopathy: raised prothrombin time
- hypoalbuminaemia
- hepatic encephalopathy
- renal failure is common ('hepatorenal syndrome')

*remember that 'liver function tests' do not always accurately reflect the synthetic function of the liver. This is best assessed by looking at the prothrombin time and albumin level.

Next question >



No SIM 10:24 PM 32%

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Questions

Lab Values, Normal Adult: Laboratory Reference Ranges in H...

Platelet count of $100 \times 10^9/l$ 17%

Body mass index of 33 kg/m^2 9%

Extrahepatic biliary obstruction 45%

With modern techniques such as ERCP and MRI cholangiography the risks of liver biopsy when there is extra-hepatic biliary obstruction are rarely justified.



 Discuss (3)
 Improve

Next question >

Liver biopsy

Contraindications to percutaneous liver biopsy

- deranged clotting (e.g. $\text{INR} > 1.4$)
- low platelets (e.g. $< 60 \times 10^9/l$)
- anaemia
- extrahepatic biliary obstruction
- hydatid cyst
- haemoangioma
- uncooperative patient
- ascites

Next question >

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Questions

The LFTs clearly show a cholestatic picture. Given the background of HIV the most likely cause is sclerosing cholangitis



Next question >

HIV: biliary and pancreatic disease

The most common cause of biliary disease in patients with HIV is sclerosing cholangitis due to infections such as CMV, Cryptosporidium and Microsporidia

Pancreatitis in the context of HIV infection may be secondary to anti-retroviral treatment (especially didanosine) or by opportunistic infections e.g. CMV

Next question >

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Search



Liver cirrhosis

Liver cirrhosis remains a significant problem in the developed world, account for 60,000 deaths in the UK each year.

Causes:

- alcohol
- non-alcoholic fatty liver disease (NAFLD)
- viral hepatitis (B and C)

Diagnosis

- traditionally a liver biopsy was used. This procedure is however associated with adverse effects such as bleeding and pain
- other techniques such as transient elastography and acoustic radiation force impulse imaging are increasingly used and were recommended by NICE in their 2016 guidelines
- for patients with NAFLD, NICE recommend using the enhanced liver fibrosis score to screen for patients who need further testing

What is transient elastography?

- brand name 'Fibroscan'
- uses a 50-MHz wave is passed into the liver from a small transducer on the end of an ultrasound probe
- measures the 'stiffness' of the liver which is a proxy for fibrosis

In terms of screening for cirrhosis NICE made a specific recommendation, suggesting to offer transient elastography to:

- people with hepatitis C virus infection
- men who drink over 50 units of alcohol per week and women who drink over 35 units of alcohol per week and have done so for several months
- people diagnosed with alcohol-related liver disease

Further investigations

- NICE recommend doing an upper endoscopy to check for varices in patient's with a new diagnosis of cirrhosis
- liver ultrasound every 6 months (+/- alpha-feto protein) to check for hepatocellular cancer

For patients with constipation who are not responding to conventional laxatives linaclotide may be considered, if:

- optimal or maximum tolerated doses of previous laxatives from different classes have not helped and
- they have had constipation for at least 12 months

Second-line pharmacological treatment

- low-dose tricyclic antidepressants (e.g. amitriptyline 5-10 mg) are used in preference to selective serotonin reuptake inhibitors

Other management options

- psychological interventions - if symptoms do not respond to pharmacological treatments after 12 months and who develop a continuing symptom profile (refractory IBS), consider referring for cognitive behavioural therapy, hypnotherapy or psychological therapy
- complementary and alternative medicines: 'do not encourage use of acupuncture or reflexology for the treatment of IBS'

General dietary advice

- have regular meals and take time to eat
- avoid missing meals or leaving long gaps between eating
- drink at least 8 cups of fluid per day, especially water or other non-caffeinated drinks such as herbal teas
- restrict tea and coffee to 3 cups per day
- reduce intake of alcohol and fizzy drinks
- consider limiting intake of high-fibre food (for example, wholemeal or high-fibre flour and breads, cereals high in bran, and whole grains such as brown rice)
- reduce intake of 'resistant starch' often found in processed foods
- limit fresh fruit to 3 portions per day
- for diarrhoea, avoid sorbitol
- for wind and bloating consider increasing intake of oats (for example, oat-based breakfast cereal or porridge) and linseeds (up to one tablespoon per day).

Next question >

Hepatic encephalopathy

Hepatic encephalopathy may be seen in liver disease of any cause. The aetiology is not fully understood but is thought to include excess absorption of ammonia and glutamine from bacterial breakdown of proteins in the gut.

Whilst hepatic encephalopathy is often associated with acute liver failure it may also be seen with chronic disease. It is now recognised that many patients with liver cirrhosis may develop subtle symptoms such as mild cognitive impairment before the features become more recognisable ('minimal' or 'covert' hepatic encephalopathy). It has also been noted that portal venous bypass with portosystemic shunting (for example post transjugular intrahepatic portosystemic shunt).

Features

- confusion, altered GCS (see below)
- asterix: 'liver flap', arrhythmic negative myoclonus with a frequency of 3-5 Hz
- constructional apraxia: inability to draw a 5-pointed star
- triphasic slow waves on EEG
- raised ammonia level (not commonly measured anymore)

Grading of hepatic encephalopathy

- Grade I: Irritability
- Grade II: Confusion, inappropriate behaviour
- Grade III: Incoherent, restless
- Grade IV: Coma

Precipitating factors

- infection e.g. spontaneous bacterial peritonitis
- GI bleed
- post transjugular intrahepatic portosystemic shunt
- constipation
- drugs: sedatives, diuretics
- hypokalaemia
- renal failure
- increased dietary protein (uncommon)

Features

- confusion, altered GCS (see below)
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- constipation
- drugs: sedatives, diuretics
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- renal failure
- increased dietary protein (uncommon)

Management

- treat any underlying precipitating cause
- NICE recommend lactulose first-line, with the addition of rifaximin for the secondary prophylaxis of hepatic encephalopathy
- lactulose is thought to work by promoting the excretion of ammonia and increasing the metabolism of ammonia by gut bacteria
- antibiotics such as rifaximin are thought to modulate the gut flora resulting in decreased ammonia production
- other options include embolisation of portosystemic shunts and liver transplantation in selected patients

Next question >

Next question >

Pyogenic liver abscess

The most common organisms found in pyogenic liver abscesses are *Staphylococcus aureus* in children and *Escherichia coli* in adults.

Management

- amoxicillin + ciprofloxacin + metronidazole
- if penicillin allergic: ciprofloxacin + clindamycin



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CT showing a pyogenic liver abscess in the right lobe of the liver.

Next question >

Next question >

Acute liver failure

Acute liver failure describes the rapid onset of hepatocellular dysfunction leading to a variety of systemic complications.

Causes

- paracetamol overdose
- alcohol
- viral hepatitis (usually A or B)
- acute fatty liver of pregnancy

Features*

- jaundice
- coagulopathy: raised prothrombin time
- hypoalbuminaemia
- hepatic encephalopathy
- renal failure is common ('hepatorenal syndrome')

*remember that 'liver function tests' do not always accurately reflect the synthetic function of the liver. This is best assessed by looking at the prothrombin time and albumin level.

Next question >

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Budd-Chiari syndrome 17%

Autoimmune hepatitis 7%

Autoimmune liver disease and alcoholic liver diseases present with AST or ALT levels below 1000. A Budd-Chiari syndrome would be visualised on the ultrasound of the abdomen. And while paracetamol toxicity can present with similarly elevated liver function tests, there needs to be a history of ingestion to make it plausible in this scenario.



Next question >

Ischaemic hepatitis

Ischaemic hepatitis is a diffuse hepatic injury resulting from acute hypoperfusion (sometimes known as 'shock liver'). It is not an inflammatory process. It is diagnosed in the presence of an inciting event (in this case a cardiac arrest) and marked increases in aminotransferase levels (exceeding 1000 international unit/L or 50 times the upper limit of normal). Often, it will occur in conjunction with acute kidney injury (tubular necrosis) or other end organ dysfunction.

Next question >

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Pregnancy: jaundice

Intrahepatic cholestasis of pregnancy

Intrahepatic cholestasis of pregnancy (also known as obstetric cholestasis) occurs in around 1% of pregnancies and is generally seen in the third trimester. It is the most common liver disease of pregnancy.

Features

- pruritus, often in the palms and soles
- no rash (although skin changes may be seen due to scratching)
- raised bilirubin

Management

- ursodeoxycholic acid is used for symptomatic relief
- weekly liver function tests
- women are typically induced at 37 weeks

Complications include an increased rate of stillbirth. It is not generally associated with increased maternal morbidity

Acute fatty liver of pregnancy

Acute fatty liver of pregnancy is rare complication which may occur in the third trimester or the period immediately following delivery.

Features

- abdominal pain
- nausea & vomiting
- headache
- jaundice
- hypoglycaemia
- severe disease may result in pre-eclampsia

Investigations

Scoring systems for liver cirrhosis

For many years the **Child-Pugh** classification was used to classify the severity of liver cirrhosis. However, in recent years the **Model for End-Stage Liver Disease (MELD)** has been increasingly used, particularly patient's who are on a liver transplant waiting list

Child-Pugh classification

Score	1	2	3
Bilirubin ($\mu\text{mol/l}$)	<34	34-50	>50
Albumin (g/l)	>35	28-35	<28
Prothrombin time, prolonged by (s)	<4	4-6	>6
Encephalopathy	none	mild	marked
Ascites	none	mild	marked

Summation of the scores allows the severity to be graded either A, B or C:

- < 7 = A
- 7-9 = B
- > 9 = C

MELD

Uses a combination of a patient's bilirubin, creatinine, and the international normalized ratio (INR) to predict survival. A formula is used to calculate the score:

$$\text{MELD} = 3.78 \times \ln[\text{serum bilirubin (mg/dL)}] + 11.2 \times \ln[\text{INR}] + 9.57 \times \ln[\text{serum creatinine (mg/dL)}] + 6.43$$

Drug-induced liver disease

Drug-induced liver disease is generally divided into hepatocellular, cholestatic or mixed. There is however considerable overlap, with some drugs causing a range of changes to the liver

The following drugs tend to cause a hepatocellular picture:

- paracetamol
- sodium valproate, phenytoin
- MAOIs
- halothane
- anti-tuberculosis: isoniazid, rifampicin, pyrazinamide
- statins
- alcohol
- amiodarone
- methyldopa
- nitrofurantoin

The following drugs tend to cause cholestasis (+/- hepatitis):

- oral contraceptive pill
- antibiotics: flucloxacillin, co-amoxiclav, erythromycin*
- anabolic steroids, testosterone
- phenothiazines: chlorpromazine, prochlorperazine
- sulphonylureas
- fibrates
- rare reported causes: nifedipine

Liver cirrhosis

- methotrexate
- methyldopa
- amiodarone

*risk may be reduced with erythromycin stearate

Next question >

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Questions

Discuss (5)

Improve

Next question >

Autoimmune hepatitis

Autoimmune hepatitis is condition of unknown aetiology which is most commonly seen in young females. Recognised associations include other autoimmune disorders, hypergammaglobulinaemia and HLA B8, DR3. Three types of autoimmune hepatitis have been characterised according to the types of circulating antibodies present

Type I	Type II	Type III
Anti-nuclear antibodies (ANA) and/or anti-smooth muscle antibodies (SMA)	Anti-liver/kidney microsomal type 1 antibodies (LKM1)	Soluble liver-kidney antigen
Affects both adults and children	Affects children only	Affects adults in middle-age

Features

- may present with signs of chronic liver disease
- acute hepatitis: fever, jaundice etc (only 25% present in this way)
- amenorrhoea (common)
- ANA/SMA/LKM1 antibodies, raised IgG levels
- liver biopsy: inflammation extending beyond limiting plate 'piecemeal necrosis', bridging necrosis

Management

- steroids, other immunosuppressants e.g. azathioprine
- liver transplantation

Next question >



Hydatid cysts



Hydatid cysts are endemic in Mediterranean and Middle Eastern countries. They are caused by the tapeworm parasite *Echinococcus granulosus*. An outer fibrous capsule is formed containing multiple small daughter cysts. These cysts are allergens which precipitate a **type 1 hypersensitivity reaction**.

Clinical features are as follows:

- Up to 90% cysts occur in the liver and lungs
- Can be asymptomatic, or symptomatic if cysts > 5cm in diameter
- Morbidity caused by cyst bursting, infection and organ dysfunction (biliary, bronchial, renal and cerebrospinal fluid outflow obstruction)
- In biliary rupture there may be the classical triad of; biliary colic, jaundice, and urticaria

CT is the best investigation to differentiate hydatid cysts from amoebic and pyogenic cysts.

Surgery is the mainstay of treatment (the cyst walls must not be ruptured during removal and the contents sterilised first).



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Questions

Lab Values, Normal Adult: Laboratory Reference Ranges in H...

Next question >

Malnutrition

Malnutrition is an important consequence of and contributor to chronic disease. It is clearly a complex and multifactorial problem that can be difficult to manage but there are a number of key points to remember for the exam.

NICE define malnutrition as the following:

- a Body Mass Index (BMI) of less than 18.5; or
- unintentional weight loss greater than 10% within the last 3-6 months; or
- a BMI of less than 20 and unintentional weight loss greater than 5% within the last 3-6 months

Around 10% of patients aged over 65 years are malnourished, the vast majority of those living independently, i.e. not in hospital or care/nursing homes.

Screening for malnutrition is mostly done using MUST (Malnutrition Universal Screen Tool). A link is provided to a copy of the MUST score algorithm.

- it should be done on admission to care/nursing homes and hospital, or if there is concern.
For example an elderly, thin patient with pressure sores
- it takes into account BMI, recent weight change and the presence of acute disease
- categorises patients into low, medium and high risk

Management of malnutrition is difficult. NICE recommend the following points:

- dietician support if the patient is high-risk
- a 'food-first' approach with clear instructions (e.g. 'add full-fat cream to mashed potato'), rather than just prescribing oral nutritional supplements (ONS) such as Ensure
- if ONS are used they should be taken between meals, rather than instead of meals

Next question >

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Refeeding syndrome

Refeeding syndrome describes the metabolic abnormalities which occur on feeding a person following a period of starvation. The metabolic consequences include:

- hypophosphataemia
- hypokalaemia
- hypomagnesaemia
- abnormal fluid balance

These abnormalities can lead to organ failure.

Prevention

NICE produced guidelines in 2006 on nutritional support. Refeeding syndrome may be avoided by identifying patients at a high-risk of developing refeeding syndrome:

Patients are considered high-risk if one or more of the following:

- BMI < 16 kg/m²
- unintentional weight loss >15% over 3-6 months
- little nutritional intake > 10 days
- hypokalaemia, hypophosphataemia or hypomagnesaemia prior to feeding (unless high)

If two or more of the following:

- BMI < 18.5 kg/m²
- unintentional weight loss > 10% over 3-6 months
- little nutritional intake > 5 days
- history of: alcohol abuse, drug therapy including insulin, chemotherapy, diuretics and antacids

NICE recommend that if a patient hasn't eaten for > 5 days, aim to re-feed at no more than 50% of requirements for the first 2 days.

Next question >

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Questions

👍 👎 Discuss (2) Improve

Next question >

Malabsorption

Malabsorption is characterised by diarrhoea, steatorrhoea and weight loss. Causes may be broadly divided into intestinal (e.g. villous atrophy), pancreatic (deficiency of pancreatic enzyme production or secretion) and biliary (deficiency of bile-salts needed for emulsification of fats)

Intestinal causes of malabsorption

- coeliac disease
- Crohn's disease
- tropical sprue
- Whipple's disease
- Giardiasis
- brush border enzyme deficiencies (e.g. lactase insufficiency)

Pancreatic causes of malabsorption

- chronic pancreatitis
- cystic fibrosis
- pancreatic cancer

Biliary causes of malabsorption

- biliary obstruction
- primary biliary cirrhosis

Other causes

- bacterial overgrowth (e.g. systemic sclerosis, diverticulæ, blind loop)
- short bowel syndrome
- lymphoma

Next question >

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Questions

Lab Values, Normal Adult: Laboratory Reference Ranges in H...

Pylorus of stomach 7%



 Discuss Improve

Next question >

VIPoma

VIP (vasoactive intestinal peptide)

- source: small intestine, pancreas
- stimulation: neural
- actions: stimulates secretion by pancreas and intestines, inhibits acid and pepsinogen secretion

VIPoma

- 90% arise from pancreas
- large volume diarrhoea
- weight loss
- dehydration
- hypokalaemia, hypochlorhydria

Next question >

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Whipple's disease

Whipple's disease is a rare multi-system disorder caused by *Tropheryma whippelii* infection. It is more common in those who are HLA-B27 positive and in middle-aged men

Features

- malabsorption: diarrhoea, weight loss
- large-joint arthralgia
- lymphadenopathy
- skin: hyperpigmentation and photosensitivity
- pleurisy, pericarditis
- neurological symptoms (rare): ophthalmoplegia, dementia, seizures, ataxia, myoclonus

Investigation

- jejunal biopsy shows deposition of macrophages containing Periodic acid-Schiff (PAS) granules

Management

- guidelines vary: oral co-trimoxazole for a year is thought to have the lowest relapse rate, sometimes preceded by a course of IV penicillin

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Question 30

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Acute pancreatitis: causes

The vast majority of cases in the UK are caused by gallstones and alcohol

Popular mnemonic is **GET SMASHED**

- Gallstones
- Ethanol
- Trauma
- Steroids
- Mumps (other viruses include Coxsackie B)
- Autoimmune (e.g. polyarteritis nodosa), *Ascaris* infection
- Scorpion venom
- Hypertriglyceridaemia, Hyperchylomicronaemia, Hypercalcaemia, Hypothermia
- ERCP
- Drugs (azathioprine, mesalazine*, didanosine, bendroflumethiazide, furosemide, pentamidine, steroids, sodium valproate)





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Question

Which one

Arthritis

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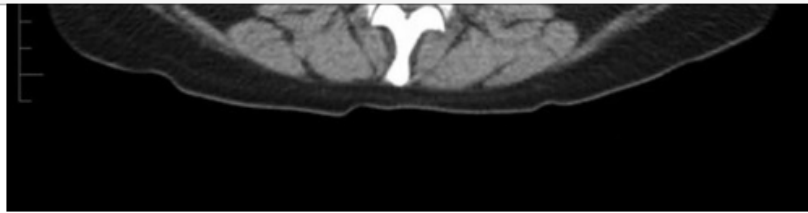
Key fac

Severe

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Abdom



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CT from a patient with acute pancreatitis. Note the diffuse parenchymal enlargement with oedema and indistinct margins.

*pancreatitis is 7 times more common in patients taking mesalazine than sulfasalazine

Rich text editor toolbar with buttons for Bold (B), Italic (I), Underline (U), Text color (A), Background color (■), Bulleted list, Numbered list, Indented list, Text color (T), Table, Image, and Link.

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Journal of Clinical and Diagnostic Research

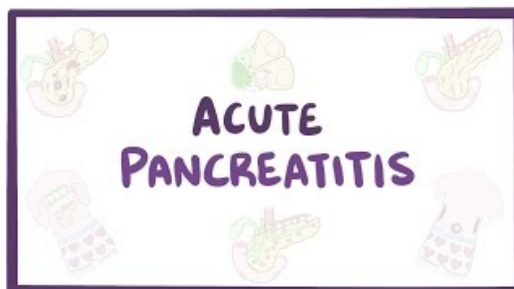
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[APACHE II scoring system for Acute Pancreatitis](#)

British Society of Gastroenterology

↑ 2 ↓ 1

[Acute pancreatitis: recent advances through randomised trials](#)



[Acute pancreatitis](#)

Osmosis - YouTube

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Questions

Lab Values, Normal Adult: Laboratory Reference Ranges in H...

ECG 12%

The cotton wool spots seen on fundoscopy represents Purtscher retinopathy. This condition may be seen following head trauma and in conditions such as acute pancreatitis, fat embolisation, amniotic fluid embolisation, and vasculitic diseases



 Discuss (5)
 Improve

Next question >

Acute pancreatitis: features

Acute pancreatitis is usually due to alcohol or gallstones.

Features:

- Severe epigastric pain that may radiate through to the back
- Vomiting is common
- Examination may reveal tenderness, ileus and low-grade fever
- Periumbilical discolouration (Cullen's sign) and flank discolouration (Grey-Turner's sign) is described but rare

Rare features associated with pancreatitis include:

- ischaemic (Purtscher) retinopathy - may cause temporary or permanent blindness

Next question >

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Chronic pancreatitis

Chronic pancreatitis is an inflammatory condition which can ultimately affect both the exocrine and endocrine functions of the pancreas. Around 80% of cases are due to alcohol excess with up to 20% of cases being unexplained

Features

- pain is typically worse 15 to 30 minutes following a meal
- steatorrhoea: symptoms of pancreatic insufficiency usually develop between 5 and 25 years after the onset of pain
- diabetes mellitus develops in the majority of patients. It typically occurs more than 20 years after symptom begin

Investigation

- abdominal x-ray shows pancreatic calcification in 30% of cases
- CT is more sensitive at detecting pancreatic calcification. Sensitivity is 80%, specificity is 85%
- functional tests: faecal elastase may be used to assess exocrine function if imaging inconclusive

Management

- pancreatic enzyme supplements
- analgesia
- antioxidants: limited evidence base - one study suggests benefit in early disease





Pancreatic cancer: features and investigation

Pancreatic cancer is often diagnosed late as it tends to present in a non-specific way. Over 80% of pancreatic tumours are adenocarcinomas which typically occur at the head of the pancreas.

Associations

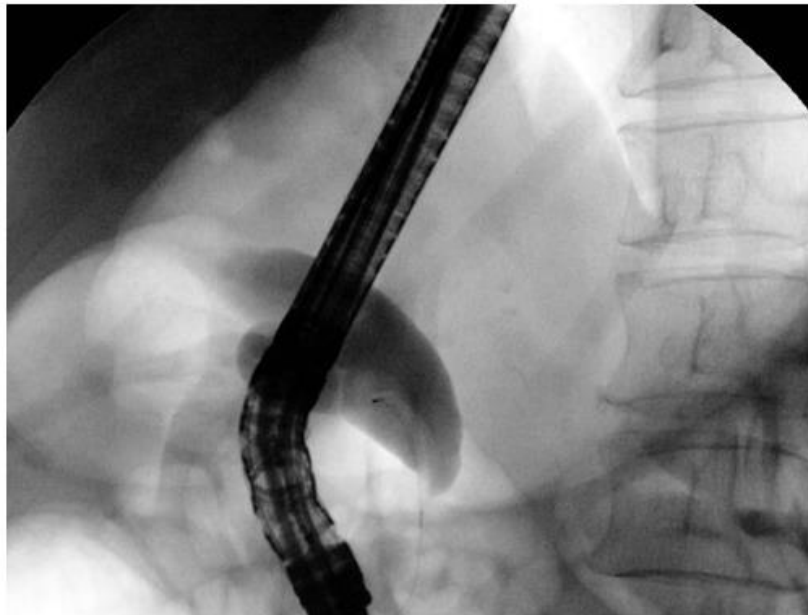
- increasing age
- smoking
- diabetes
- chronic pancreatitis (alcohol does not appear an independent risk factor though)
- hereditary non-polyposis colorectal carcinoma
- multiple endocrine neoplasia
- BRCA2 gene

Features

- classically painless jaundice
- however, patients typically present in a non-specific way with anorexia, weight loss, epigastric pain
- loss of exocrine function (e.g. steatorrhoea)
- atypical back pain is often seen
- migratory thrombophlebitis (Trousseau sign) is more common than with other cancers

Investigation

- ultrasound has a sensitivity of around 60-90%
- high resolution CT scanning is the investigation of choice if the diagnosis is suspected



Next question >

Zollinger-Ellison syndrome

Zollinger-Ellison syndrome is condition characterised by excessive levels of gastrin, usually from a gastrin secreting tumour usually of the duodenum or pancreas. Around 30% occur as part of MEN type I syndrome

Features

- multiple gastroduodenal ulcers
- diarrhoea
- malabsorption

Diagnosis

- fasting gastrin levels: the single best screen test
- secretin stimulation test

Next question >

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Dysphagia



The table below gives characteristic exam question features for conditions causing dysphagia:

Causes	Notes
Oesophageal cancer	Dysphagia may be associated with weight loss, anorexia or vomiting during eating Past history may include Barrett's oesophagus, GORD, excessive smoking or alcohol use
Oesophagitis	May be history of heartburn Odynophagia but no weight loss and systemically well
Oesophageal candidiasis	There may be a history of HIV or other risk factors such as steroid inhaler use
Achalasia	Dysphagia of both liquids and solids from the start Heartburn Regurgitation of food - may lead to cough, aspiration pneumonia etc
Pharyngeal pouch	More common in older men Represents a posteromedial herniation between thyropharyngeus and cricopharyngeus muscles Usually not seen but if large then a midline lump in the neck that gurgles on palpation Typical symptoms are dysphagia, regurgitation, aspiration and chronic cough. Halitosis may occasionally be seen
Systemic sclerosis	Other features of CREST syndrome may be present, namely Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia As well as oesophageal dysmotility the lower oesophageal sphincter (LES) pressure is decreased. This contrasts to achalasia where the LES pressure is increased
Myasthenia gravis	Other symptoms may include extraocular muscle weakness or ptosis Dysphagia with liquids as well as solids
Globus hystericus	May be history of anxiety Symptoms are often intermittent and relieved by swallowing Usually painless - the presence of pain should warrant further investigation for organic causes

Usually painless - the presence of pain should warrant further investigation for organic causes

Causes of dysphagia - by classification

As with many conditions, it's often useful to think about causes of a symptom in a structured way:

Classification	Examples
Extrinsic	• Mediastinal masses • Cervical spondylosis
Oesophageal wall	• Achalasia • Diffuse oesophageal spasm • Hypertensive lower oesophageal sphincter
Intrinsic	• Tumours • Strictures • Oesophageal web • Schatzki rings
Neurological	• CVA • Parkinson's disease • Multiple Sclerosis • Brainstem pathology • Myasthenia Gravis

Investigation

All patients require an upper GI endoscopy unless there are compelling reasons for this not to be performed. Motility disorders may be best appreciated by undertaking fluoroscopic swallowing studies.

A full blood count should be performed.

Ambulatory oesophageal pH and manometry studies will be required to evaluate conditions such as achalasia and patients with GORD being considered for fundoplication surgery.

Question 12

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Middle aged female, fatigue, pruritus, raised IgM - primary biliary cirrhosis

Notes (1/1)

Oesophageal disorders

The table below lists a small group of oesophageal disorders that are not covered elsewhere in the notes.

Disorder	Notes
Plummer-Vinson syndrome	Triad of: - dysphagia (secondary to oesophageal webs) - glossitis - iron-deficiency anaemia Treatment includes iron supplementation and dilation of the webs
Mallory-Weiss syndrome	Severe vomiting → painful mucousal lacerations at the gastroesophageal junction resulting in haematemesis. Common in alcoholics
Boerhaave syndrome	Severe vomiting → oesophageal rupture

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Close

Achalasia

Failure of oesophageal peristalsis and of relaxation of lower oesophageal sphincter (LOS) due to degenerative loss of ganglia from Auerbach's plexus i.e. LOS contracted, oesophagus above dilated. Achalasia typically presents in middle-age and is equally common in men and women.

Clinical features

- dysphagia of BOTH liquids and solids
- typically variation in severity of symptoms
- heartburn
- regurgitation of food - may lead to cough, aspiration pneumonia etc
- malignant change in small number of patients

Investigations

- manometry: excessive LOS tone which doesn't relax on swallowing - considered most important diagnostic test
- barium swallow shows grossly expanded oesophagus, fluid level, 'bird's beak' appearance
- CXR: wide mediastinum, fluid level

Treatment

- intra-sphincteric injection of botulinum toxin
- Heller cardiomyotomy
- balloon dilation
- drug therapy has a role but is limited by side-effects



Barrett's oesophagus

Barrett's refers to the metaplasia of the lower oesophageal mucosa, with the usual squamous epithelium being replaced by columnar epithelium. There is an increased risk of oesophageal adenocarcinoma, estimated at 50-100 fold. There are no screening programs for Barrett's - it's typically identified when patients have an endoscopy for evaluation of upper gastrointestinal symptoms such as dyspepsia.

Barrett's can be subdivided into short (<3cm) and long (>3cm). The length of the affected segment correlates strongly with the chances of identifying metaplasia. The overall prevalence of Barrett's oesophagus is difficult to determine but may be in the region of 1 in 20 and is identified in up to 12% of those undergoing endoscopy for reflux.

Histological features

- the columnar epithelium may resemble that of either the cardiac region of the stomach or that of the small intestine (e.g. with goblet cells, brush border)

Risk factors

- gastro-oesophageal reflux disease (GORD) is the single strongest risk factor
- male gender (7:1 ratio)
- smoking
- central obesity

Interestingly alcohol does not seem to be an independent risk factor for Barrett's although it is associated with both GORD and oesophageal cancer.

Whilst Barrett's oesophagus itself is asymptomatic clearly patients will often have coexistent GORD symptoms.

Management

- endoscopic surveillance with biopsies
- high-dose proton pump inhibitor: whilst this is commonly used in patients with Barrett's the evidence base that this reduces the change of progression to dysplasia or induces regression of the lesion is limited

Endoscopic surveillance

Management

- endoscopic surveillance with biopsies
- high-dose proton pump inhibitor: whilst this is commonly used in patients with Barrett's the evidence base that this reduces the change of progression to dysplasia or induces regression of the lesion is limited

Endoscopic surveillance

- for patients with metaplasia (but not dysplasia) endoscopy is recommended every 3-5 years

If dysplasia of any grade is identified endoscopic intervention is offered. Options include:

- endoscopic mucosal resection
- radiofrequency ablation



Endoscopy image showing a short segment of Barrett's oesophagus

Next question >

Oesophageal cancer

Until recent times oesophageal cancer was most commonly due to a squamous cell carcinoma but the incidence of adenocarcinoma is rising rapidly. Adenocarcinoma is now the most common type of oesophageal cancer and is more likely to develop in patients with a history of gastro-oesophageal reflux disease (GORD) or Barrett's.

The majority of tumours are in the middle third of the oesophagus.

Risk factors

- smoking
- alcohol
- GORD
- Barrett's oesophagus
- achalasia
- Plummer-Vinson syndrome
- squamous cell carcinoma is also linked to diets rich in nitrosamines
- rare: coeliac disease, scleroderma

Features

- dysphagia: the most common presenting symptom
- anorexia and weight loss
- vomiting
- other possible features include: odynophagia, hoarseness, melaena, cough

Diagnosis

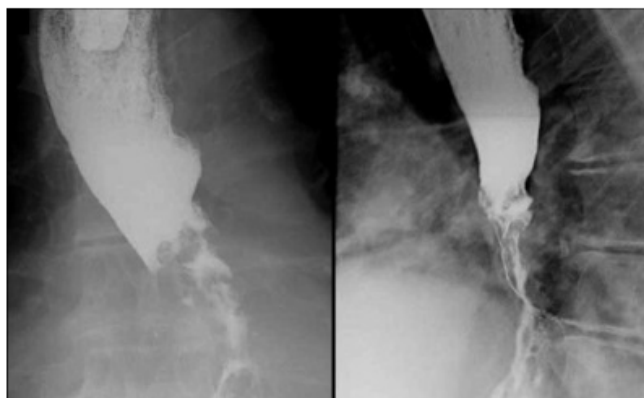
- Upper GI endoscopy is the first line test
- Contrast swallow may be of benefit in classifying benign motility disorders but has no place in the assessment of tumours
- Staging is initially undertaken with CT scanning of the chest, abdomen and pelvis. If overt metastatic disease is identified using this modality then further complex imaging is unnecessary
- If CT does not show metastatic disease, then local stage may be more accurately assessed by use of endoscopic ultrasound.
- Staging laparoscopy is performed to detect occult peritoneal disease. PET CT is performed in those with negative laparoscopy. Thoracoscopy is not routinely performed.

Diagnosis

- Upper GI endoscopy is the first line test
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Treatment

- Operable disease is best managed by surgical resection.
- The most standard procedure is an Ivor- Lewis type oesophagectomy. This procedure involves the mobilisation of the stomach and division of the oesophageal hiatus. The abdomen is closed and a right sided thoracotomy performed. The stomach is brought into the chest and the oesophagus mobilised further. An intrathoracic oesophagogastric anastomosis is constructed. Alternative surgical strategies include a transhiatal resection (for distal lesions), a left thoraco-abdominal resection (difficult access due to thoracic aorta) and a total oesophagectomy (McKeown) with a cervical oesophagogastric anastomosis.
- The biggest surgical challenge is that of anastomotic leak, with an intrathoracic anastomosis this will result in mediastinitis. With high mortality. The McKeown technique has an intrinsically lower systemic insult in the event of anastomotic leakage.
- In addition to surgical resection many patients will be treated with adjuvant chemotherapy.



Dyspepsia

The 2015 NICE guidelines 'Suspected cancer: recognition and referral' further updated the advice on who needs urgent referral for an endoscopy (i.e. within 2 weeks). The list below combines the advice for oesophageal and stomach cancer, with the bold added by the author, not NICE.

Urgent

All patients who've got **dysphagia**

All patients who've got an **upper abdominal mass** consistent with stomach cancer

Patients aged ≥ 55 years who've got **weight loss**, AND any of the following:

- upper abdominal pain
- reflux
- dyspepsia

Non-urgent

Patients with **haematemesis**

Patients aged ≥ 55 years who've got:

- **treatment-resistant dyspepsia** or
- upper abdominal pain with low haemoglobin levels or
- **raised platelet count** with any of the following: nausea, vomiting, weight loss, reflux, dyspepsia, upper abdominal pain
- nausea or vomiting with any of the following: weight loss, reflux, dyspepsia, upper abdominal pain

Managing patients who do not meet referral criteria ('undiagnosed dyspepsia')

This can be summarised at a step-wise approach

- 1. Review medications for possible causes of dyspepsia
- 2. Lifestyle advice
- 3. Trial of full-dose proton pump inhibitor for one month OR a 'test and treat' approach for

- reflux
- dyspepsia

Non-urgent

Patients with **haematemesis**

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Managing patients who do not meet referral criteria ('undiagnosed dyspepsia')

This can be summarised at a step-wise approach

1. Review medications for possible causes of dyspepsia
2. Lifestyle advice
3. Trial of full-dose proton pump inhibitor for one month OR a 'test and treat' approach for *H. pylori*

Testing for *H. pylori* infection

- initial diagnosis: NICE recommend using a carbon-13 urea breath test or a stool antigen test, or laboratory-based serology 'where its performance has been locally validated'
- test of cure: carbon-13 urea breath test

Next question >

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Variceal haemorrhage

Acute treatment of variceal haemorrhage

- ABC: patients should ideally be resuscitated prior to endoscopy
- correct clotting: FFP, vitamin K
- vasoactive agents: terlipressin is currently the only licensed vasoactive agent and is supported by NICE guidelines. It has been shown to be of benefit in initial haemostasis and preventing rebleeding. Octreotide may also be used although there is some evidence that terlipressin has a greater effect on reducing mortality
- prophylactic antibiotics have been shown to reduce mortality in patients with liver cirrhosis. Quinolones are typically used. NICE support this in their 2016 guidelines: *'Offer prophylactic intravenous antibiotics for people with cirrhosis who have upper gastrointestinal bleeding.'*
- endoscopy: endoscopic variceal band ligation is superior to endoscopic sclerotherapy. NICE recommend band ligation
- Sengstaken-Blakemore tube if uncontrolled haemorrhage
- Transjugular Intrahepatic Portosystemic Shunt (TIPSS) if above measures fail

Prophylaxis of variceal haemorrhage

- propranolol: reduced rebleeding and mortality compared to placebo
- endoscopic variceal band ligation (EVL) is superior to endoscopic sclerotherapy. It should be performed at two-weekly intervals until all varices have been eradicated. Proton pump inhibitor cover is given to prevent EVL-induced ulceration. This is supported by NICE who recommend: *'Offer endoscopic variceal band ligation for the primary prevention of bleeding for people with cirrhosis who have medium to large oesophageal varices.'*

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NICE

3 0

2012 Acute upper gastrointestinal bleeding: management

Royal College of Physicians

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2012 Managing acute upper gastrointestinal bleeding in the acute assessment unit

Acute upper gastrointestinal bleeding

NICE published guidelines in 2012 on the management of acute upper gastrointestinal bleeding which is most commonly due to either peptic ulcer disease or oesophageal varices. Some of the key points are detailed below.

Risk assessment

- use the Blatchford score at first assessment, and
- the full Rockall score after endoscopy

Blatchford score

Admission risk marker	Score
Urea (mmol/l)	6.5 - 8 = 2 8 - 10 = 3 10 - 25 = 4 > 25 = 6
Haemoglobin (g/l)	Men • 12 - 13 = 1 • 10 - 12 = 3 • < 10 = 6 Women • 10 - 12 = 1 • < 10 = 6
Systolic blood pressure (mmHg)	100 - 109 = 1 90 - 99 = 2 < 90 = 3
Other markers	Pulse >=100/min = 1 Presentation with melaena = 1 Presentation with syncope = 2

No SIM 2:18 PM 90% Systolic blood pressure (mmHg) passmedicine.com 90 - 99 = 2 < 80 = 3 Lab Values, Normal Adult: Laboratory Reference Ranges in H...	
Questions	
Other markers	Pulse $\geq 100/\text{min}$ = 1 Presentation with melaena = 1 Presentation with syncope = 2 Hepatic disease = 2 Cardiac failure = 2

Patients with a Blatchford score of 0 may be considered for early discharge

Resuscitation

- ABC, wide-bore intravenous access * 2
- platelet transfusion if actively bleeding platelet count of less than $50 \times 10^9/\text{litre}$
- fresh frozen plasma to patients who have either a fibrinogen level of less than 1 g/litre, or a prothrombin time (international normalised ratio) or activated partial thromboplastin time greater than 1.5 times normal
- prothrombin complex concentrate to patients who are taking warfarin and actively bleeding

Endoscopy

- should be offered immediately after resuscitation in patients with a severe bleed
- all patients should have endoscopy within 24 hours

Management of non-variceal bleeding

- NICE do not recommend the use of proton pump inhibitors (PPIs) before endoscopy to patients with suspected non-variceal upper gastrointestinal bleeding although PPIs should be given to patients with non-variceal upper gastrointestinal bleeding and stigmata of recent haemorrhage shown at endoscopy
- if further bleeding then options include repeat endoscopy, interventional radiology and surgery

Management of variceal bleeding

- terlipressin and prophylactic antibiotics should be given to patients at presentation (i.e. before endoscopy)
- band ligation should be used for oesophageal varices and injections of N-butyl-2-cyanoacrylate for patients with gastric varices
- transjugular intrahepatic portosystemic shunts (TIPS) should be offered if bleeding from

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Questions

- fresh frozen plasma to patients who have either a fibrinogen level of less than 1 g/litre, or a prothrombin time (international normalised ratio) or activated partial thromboplastin time greater than 1.5 times normal
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- transjugular intrahepatic portosystemic shunts (TIPS) should be offered if bleeding from varices is not controlled with the above measures

Next question >

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Questions

Lab Values, Normal Adult: Laboratory Reference Ranges in H...

Barium swallow 4%

CT thorax 2%

24hr oesophageal pH monitoring is gold standard investigation in GORD

👍 👎 Discuss Improve

Next question >

Gastro-oesophageal reflux disease: investigation

Overview

- poor correlation between symptoms and endoscopy appearance

Indications for upper GI endoscopy:

- age > 55 years
- symptoms > 4 weeks or persistent symptoms despite treatment
- dysphagia
- relapsing symptoms
- weight loss

If endoscopy is negative consider 24-hr oesophageal pH monitoring (the gold standard test for diagnosis)

Next question >

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Helicobacter pylori



Helicobacter pylori is a Gram negative bacteria associated with a variety of gastrointestinal problems, principally peptic ulcer disease

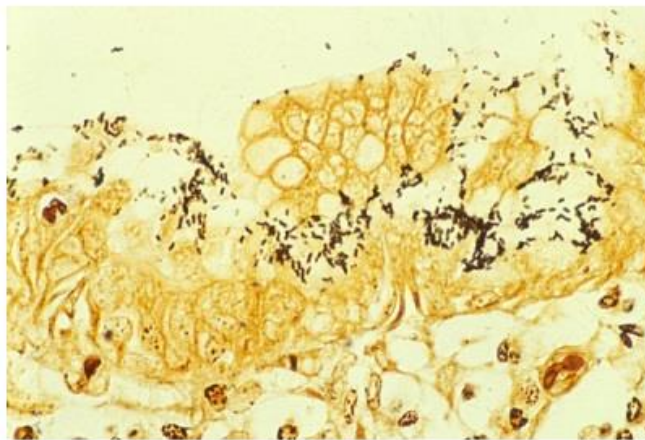
Associations

- peptic ulcer disease (95% of duodenal ulcers, 75% of gastric ulcers)
- gastric cancer
- B cell lymphoma of MALT tissue (eradication of H pylori results causes regression in 80% of patients)
- atrophic gastritis

The role of H pylori in Gastro-oesophageal reflux disease (GORD) is unclear - there is currently no role in GORD for the eradication of H pylori

Management - eradication may be achieved with a 7 day course of

- a proton pump inhibitor + amoxicillin + clarithromycin, or
- a proton pump inhibitor + metronidazole + clarithromycin



H. pylori colonized on the surface of regenerative epithelium (silver stain)

B I U

Helicobacter pylori: tests

Urea breath test

- patients consume a drink containing carbon isotope 13 (^{13}C) enriched urea
- urea is broken down by *H. pylori* urease
- after 30 mins patient exhale into a glass tube
- mass spectrometry analysis calculates the amount of ^{13}C CO_2
- should not be performed within 4 weeks of treatment with an antibacterial or within 2 weeks of an antisecretory drug (e.g. a proton pump inhibitor)
- sensitivity 95-98%, specificity 97-98%

Rapid urease test (e.g. CLO test)

- biopsy sample is mixed with urea and pH indicator
- colour change if *H. pylori* urease activity
- sensitivity 90-95%, specificity 95-98%

Serum antibody

- remains positive after eradication
- sensitivity 85%, specificity 80%

Culture of gastric biopsy

- provide information on antibiotic sensitivity
- sensitivity 70%, specificity 100%

Gastric biopsy

- histological evaluation alone, no culture
- sensitivity 95-99%, specificity 95-99%

Stool antigen test

- sensitivity 90%, specificity 95%

Next question >

Relevant to my revision >

Less relevant >

Proton pump inhibitors

Proton pump inhibitors (PPI) are a group of drugs which profoundly reduce acid secretion in the stomach. They irreversibly blocking the hydrogen/potassium adenosine triphosphatase enzyme system (the H^+/K^+ ATPase) of the gastric parietal cell

Examples include omeprazole and lansoprazole

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84 facts found

Score for this session: **86.2%**

Key facts from this session are shown below (score/attempts), click on the fact for background information

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Questions

Lab Values, Normal Adult: Laboratory Reference Ranges in H...

Jejunal mucosa antibodies	3%
Intrinsic factor antibodies	48%
Vitamin B12 antibodies	3%



 Discuss (1) Improve

Next question >

Pernicious anaemia: investigation

Investigation

- anti gastric parietal cell antibodies in 90% (but low specificity)
- anti intrinsic factor antibodies in 50% (specific for pernicious anaemia)
- macrocytic anaemia
- low WCC and platelets
- LDH may be raised due to ineffective erythropoiesis
- also low serum B12, hypersegmented polymorphs on film, megaloblasts in marrow
- Schilling test

Schilling test

- radiolabelled B12 given on two occasions
- first on its own
- second with oral IF
- urine B12 levels measured

Next question >

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Gastric cancer

There are 700,000 new cases of gastric cancer worldwide each year.

Epidemiology

- overall incidence is decreasing, but incidence of tumours arising from the cardia is increasing
- peak age = 70-80 years
- more common in Japan, China, Finland and Colombia than the West
- more common in males, 2:1

Histology

- signet ring cells may be seen in gastric cancer. They contain a large vacuole of mucin which displaces the nucleus to one side. Higher numbers of signet ring cells are associated with a worse prognosis

Associations

- *H. pylori* infection
- blood group A: gAstric cAncer
- gastric adenomatous polyps
- pernicious anaemia
- smoking
- diet: salty, spicy, nitrates
- may be negatively associated with duodenal ulcer

Features

- dyspepsia
- nausea and vomiting
- anorexia and weight loss
- dysphagia

Investigation

- diagnosis: endoscopy with biopsy
- staging: CT or endoscopic ultrasound - endoscopic ultrasound has recently been shown to be superior to CT

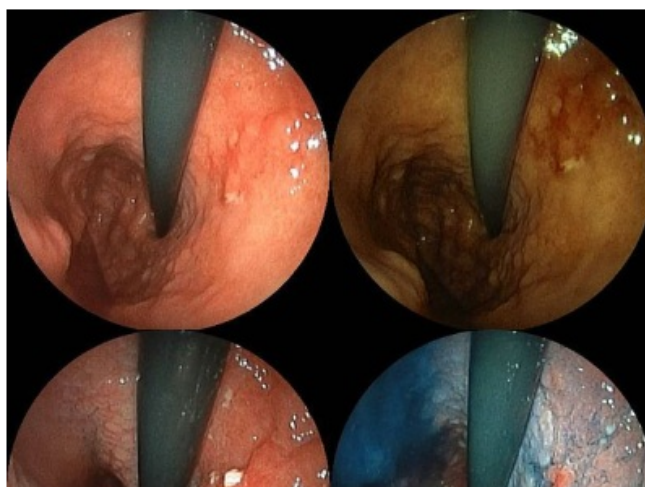
Investigation

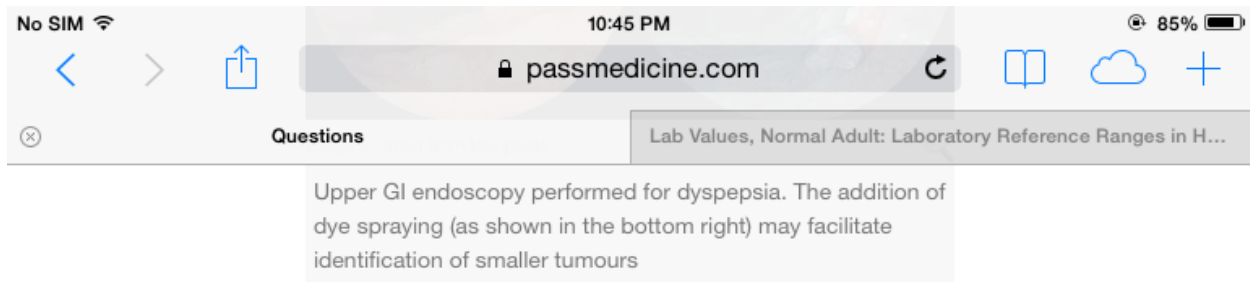
- diagnosis: endoscopy with biopsy
- staging: CT or endoscopic ultrasound - endoscopic ultrasound has recently been shown to be superior to CT

Surgical aspects

There is some evidence of support a stepwise progression of the disease through intestinal metaplasia progressing to atrophic gastritis and subsequent dysplasia, through to cancer. The favoured staging system is TNM. The risk of lymph node involvement is related to size and depth of invasion; early cancers confined to submucosa have a 20% incidence of lymph node metastasis. Tumours of the gastro-oesophageal junction are classified as below:

Type 1	True oesophageal cancers and may be associated with Barrett's oesophagus.
Type 2	Carcinoma of the cardia, arising from cardiac type epithelium or short segments with intestinal metaplasia at the oesophagogastric junction.
Type 3	Sub cardial cancers that spread across the junction. Involve similar nodal stations to gastric cancer.





Staging

- CT scanning of the chest abdomen and pelvis is the routine first line staging investigation in most centres.
- Laparoscopy to identify occult peritoneal disease
- PET CT (particularly for junctional tumours)

Treatment

- Proximally sited disease greater than 5-10cm from the OG junction may be treated by sub total gastrectomy
- Total gastrectomy if tumour is <5cm from OG junction
- For type 2 junctional tumours (extending into oesophagus) oesophagogastrrectomy is usual
- Endoscopic sub mucosal resection may play a role in early gastric cancer confined to the mucosa and perhaps the sub mucosa (this is debated)
- Lymphadenectomy should be performed. A D2 lymphadenectomy is widely advocated by the Japanese, the survival advantages of extended lymphadenectomy have been debated. However, the overall recommendation is that a D2 nodal dissection be undertaken.
- Most patients will receive chemotherapy either pre or post operatively.

Prognosis

UK Data

Disease extent	Percentage 5 year survival
All RO resections	54%
Early gastric cancer	91%
Stage 1	87%
Stage 2	65%

Prognosis

UK Data

Disease extent	Percentage 5 year survival
All RO resections	54%
Early gastric cancer	91%
Stage 1	87%
Stage 2	65%
Stage 3	18%

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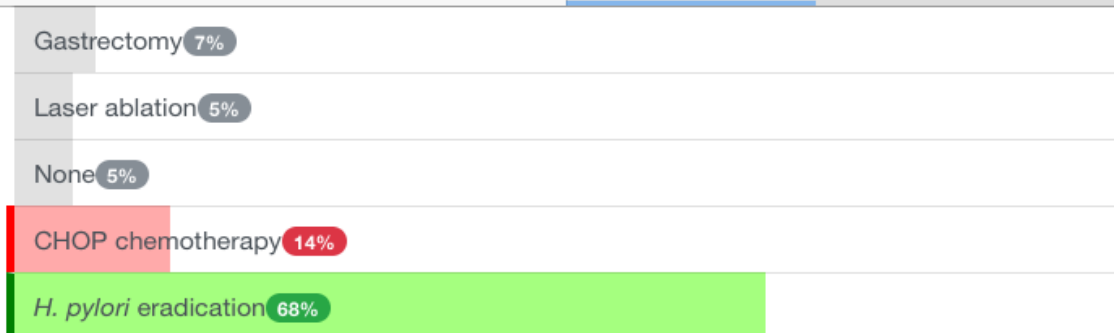
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Gastric MALT lymphoma - eradicate *H. pylori*

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Next question >

Gastric MALT lymphoma

Overview

- associated with *H. pylori* infection in 95% of cases
- good prognosis
- if low grade then 80% respond to *H. pylori* eradication

Features

- paraproteinaemia may be present

Next question >

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Questions
Tetracyclines are no longer commonly used

Discuss (1) Improve

Next question >

Small bowel bacterial overgrowth syndrome

Small bowel bacterial overgrowth syndrome (SBBOS) is a disorder characterised by excessive amounts of bacteria in the small bowel resulting in gastrointestinal symptoms.

Risk factors for SBBOS

- neonates with congenital gastrointestinal abnormalities
- scleroderma
- diabetes mellitus

It should be noted that many of the features overlap with irritable bowel syndrome:

- chronic diarrhoea
- bloating, flatulence
- abdominal pain

Diagnosis

- hydrogen breath test

Management

- correction of underlying disorder
- antibiotic therapy: rifaximin is now the treatment of choice due to relatively low resistance. Co-amoxiclav or metronidazole are also effective in the majority of patients.

Next question >

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The hydrogen breath test is another option. It has low sensitivity but high specificity

  Discuss (1) Improve

Next question >

Bacterial overgrowth: investigation

The gold standard investigation of bacterial overgrowth is small bowel aspiration and culture

Other possible investigations include:

- hydrogen breath test
- 14C-xylose breath test
- 14C-glycocholate breath test: used increasingly less due to low specificity

In practice many clinicians give an empirical course of antibiotics as a trial

Next question >

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Villous adenoma

Villous adenomas are colonic polyps with the potential for malignant transformation. They characteristically secrete large amounts of mucous, potentially resulting in electrolyte disturbances.

The vast majority are asymptomatic. Possible features:

- non-specific lower gastrointestinal symptoms
- secretory diarrhoea may occur
- microcytic anaemia
- hypokalaemia

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Close

135 facts

Score for this session: 75.6%

7 in a row correct

Key facts from this session are shown below (score/attempts), click on the fact for background information

A 75-year-old man presents with passing large amounts of a mucous-like diarrhoea. Bloods demonstrate hypokalaemia - villous adenoma

[Notes](#) (1/1)

A young man is noted to have an elevated unconjugated bilirubin level on a fasting blood sample - Gilbert's syndrome

[Notes](#) (1/1)

Diverticulosis

Diverticulosis is an extremely common disorder characterised by multiple outpouchings of the bowel wall, most commonly in the sigmoid colon. Strictly speaking the term diverticular disease is reserved for patients who are symptomatic - diverticulosis is the more accurate term for diverticula being present.

Diverticulosis can present in a number of ways:

- painful diverticular disease: altered bowel habit, colicky left sided abdominal pain. A high fibre diet is usually recommended to minimise symptoms
- diverticulitis: see below for more details

Diverticulitis

One of the diverticular become infected. The classical presentation is:

- left iliac fossa pain and tenderness
- anorexia, nausea and vomiting
- diarrhoea
- features of infection (pyrexia, raised WBC and CRP)

Management:

- mild attacks can be treated with oral antibiotics
- more significant episodes are managed in hospital. Patients are made nil by mouth, intravenous fluids and intravenous antibiotics (typical a cephalosporin + metronidazole) are given

Complications of diverticulitis include:

- abscess formation
- peritonitis
- obstruction
- perforation

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Question

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29 facts

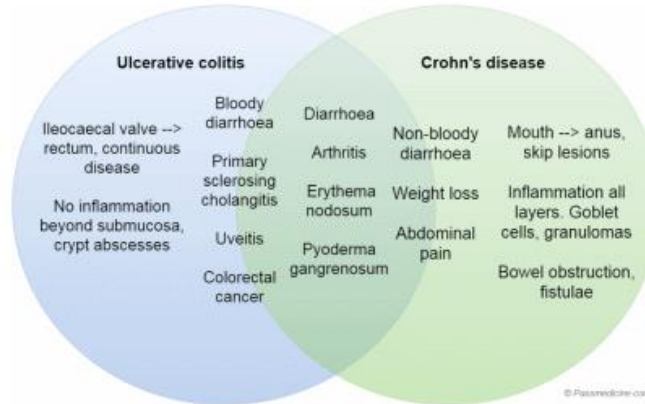
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Key facts

A 20-year-old
colitisA 50-year-old
examined
ultrasound

Session



Venn diagram showing shared features and differences between ulcerative colitis and Crohn's disease. Note that whilst some features are present in both, some are much more common in one of the conditions, for example colorectal cancer in ulcerative colitis

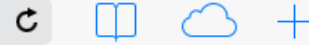
Pathology

- red, raw mucosa, bleeds easily
- no inflammation beyond submucosa (unless fulminant disease)
- widespread ulceration with preservation of adjacent mucosa which has the appearance of polyps ('pseudopolyps')
- inflammatory cell infiltrate in lamina propria
- neutrophils migrate through the walls of glands to form crypt abscesses
- depletion of goblet cells and mucin from gland epithelium
- granulomas are infrequent

Barium enema

- loss of haustrations
- superficial ulceration, 'pseudopolyps'
- long standing disease: colon is narrow and short - 'drainpipe colon'





Acute appendicitis



Acute appendicitis is the most common acute abdominal condition requiring surgery. It can occur at any age but is most common in young people aged 10-20 years.

Abdominal pain is seen in the vast majority of patients:

- peri-umbilical abdominal pain (visceral stretching of appendix lumen and appendix is midgut structure) radiating to the right iliac fossa (RIF) due to localised parietal peritoneal inflammation.
- the migration of the pain from the centre to the RIF has been shown to be one of the strongest indicators of appendicitis
- patients often report the pain being worse on coughing or going over speed bumps. Children typically can't hop on the right leg due to the pain.

Other features:

- vomit once or twice but marked and persistent vomiting is unusual
- diarrhoea is rare. However, pelvic appendicitis may cause localised rectal irritation of some loose stools. A pelvic abscess may also cause diarrhoea
- mild pyrexia is common - temperature is usually 37.5-38°C. Higher temperatures are more typical of conditions like mesenteric adenitis
- anorexia is very common. It is very unusual for patients with appendicitis to be hungry
- around 50% of patients have the typical symptoms of anorexia, peri-umbilical pain and nausea followed by more localised right lower quadrant pain

Examination

- generalised peritonitis if perforation has occurred or localised peritonism
- retrocaecal appendicitis may have relatively few signs
- digital rectal examination may reveal boggy sensation if pelvic abscess is present, or even tenderness with a pelvic appendix
- Rovsing's sign (palpation in the LIF causes pain in the RIF) is now thought to be of limited value

Diagnosis

- typically raised inflammatory markers coupled with compatible history and examination findings should be enough to justify appendicectomy.
- urine analysis: useful to exclude pregnancy in women, renal colic and urinary tract infection. In patients with appendicitis, urinalysis may show mild leucocytosis but no nitrites
- ultrasound is useful in females where pelvic organ pathology is suspected. Although it is not always possible to visualise the appendix on ultrasound, the presence of free fluid (always pathological in males) should raise suspicion
- CT scans are widely used in patients with suspected appendicitis in the US but this practice has not currently reached the UK due to the concerns regarding excessive



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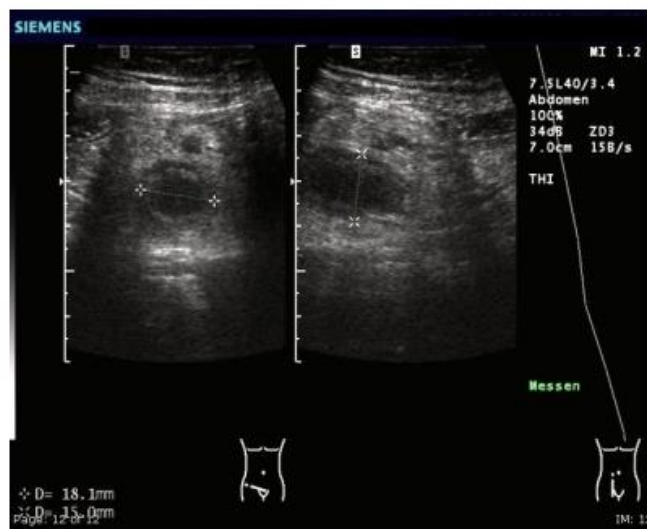
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Diagnosis

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Management

- appendicectomy which can be performed via either an open or laparoscopic approach. Laparoscopic appendicectomy is now the treatment of choice
- administration of metronidazole reduces wound infection rates.
- patients with perforated appendicitis (typical around 15-20%) require copious abdominal lavage.
- patients without peritonitis who have an appendix mass should receive broad-spectrum antibiotics and consideration given to performing an interval appendicectomy.
- be wary in the older patients who may have either an underlying caecal malignancy or perforated sigmoid diverticular disease.
- trials have looked at the use of intravenous antibiotics alone in the treatment of appendicitis. The evidence currently suggests that whilst this is successful in the majority of patients, it is associated with a longer hospital stay and up to 20% of patients go on to have an appendicectomy within 12 months.





Next question >

Jejunal villous atrophy

Whilst coeliac disease is the classic cause of jejunal villous atrophy there are a number of other causes you need to be aware of

Causes

- coeliac disease
- tropical sprue
- hypogammaglobulinaemia
- gastrointestinal lymphoma
- Whipple's disease
- cow's milk intolerance

Next question >

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Next question >

Anal fissure

Anal fissures are longitudinal or elliptical tears of the squamous lining of the distal anal canal. If present for less than 6 weeks they are defined as acute, and chronic if present for more than 6 weeks. Around 90% of anal fissures occur on the posterior midline

Risk factors

- constipation
- inflammatory bowel disease
- sexually transmitted infections e.g. HIV, syphilis, herpes

Features

- painful, bright red, rectal bleeding

Management of an acute anal fissure (< 6 weeks)

- dietary advice: high-fibre diet with high fluid intake
- bulk-forming laxatives are first line - if not tolerated then lactulose should be tried
- lubricants such as petroleum jelly may be tried before defecation
- topical anaesthetics

-analgesia

- topical steroids do not provide significant relief

Management of a chronic anal fissure (> 6 weeks)

- the above techniques should be continued
- topical glyceryl trinitrate (GTN) is first line treatment for a chronic anal fissure
- if topical GTN is not effective after 8 weeks then secondary referral should be considered for surgery or botulinum toxin

Next question >

Next question >

Colorectal cancer: referral guidelines

NICE updated their referral guidelines in 2015. The following patients should be referred urgently (i.e. within 2 weeks) to colorectal services for investigation:

- patients ≥ 40 years with unexplained weight loss **AND** abdominal pain
- patients ≥ 50 years with unexplained rectal bleeding
- patients ≥ 60 years with iron deficiency anaemia **OR** change in bowel habit
- tests show occult blood in their faeces (see below)

An urgent referral (within 2 weeks) should be 'considered' if:

- there is a rectal or abdominal mass
- there is an unexplained anal mass or anal ulceration
- patients < 50 years with rectal bleeding **AND** any of the following unexplained symptoms/findings:
 - $\rightarrow$ abdominal pain
 - $\rightarrow$ change in bowel habit
 - $\rightarrow$ weight loss
 - $\rightarrow$ iron deficiency anaemia

Faecal Occult Blood Testing (FOBT)

This was one of the main changes in 2015. Remember that the NHS now has a national screening programme offering screening every 2 years to all men and women aged 60 to 74 years. Patients aged over 74 years may request screening.

In addition FOBT should be offered to:

- patients ≥ 50 years with unexplained abdominal pain **OR** weight loss
- patients < 60 years with changes in their bowel habit **OR** iron deficiency anaemia
- patients ≥ 60 years who have anaemia even in the absence of iron deficiency

Next question >

Colorectal cancer: genetics

It is currently thought there are three types of colon cancer:

- sporadic (95%)
- hereditary non-polyposis colorectal carcinoma (HNPCC, 5%)
- familial adenomatous polyposis (FAP, <1%)

Studies have shown that sporadic colon cancer may be due to a series of genetic mutations. For example, more than half of colon cancers show allelic loss of the APC gene. It is believed a further series of gene abnormalities e.g. activation of the K-ras oncogene, deletion of p53 and DCC tumour suppressor genes lead to invasive carcinoma

HNPCC, an autosomal dominant condition, is the most common form of inherited colon cancer. Around 90% of patients develop cancers, often of the proximal colon, which are usually poorly differentiated and highly aggressive. Currently seven mutations have been identified, which affect genes involved in DNA mismatch repair leading to microsatellite instability. The most common genes involved are:

- MSH2 (60% of cases)
- MLH1 (30%)

Patients with HNPCC are also at a higher risk of other cancers, with endometrial cancer being the next most common association, after colon cancer.

The Amsterdam criteria are sometimes used to aid diagnosis:

- at least 3 family members with colon cancer
- the cases span at least two generations
- at least one case diagnosed before the age of 50 years

FAP is a rare autosomal dominant condition which leads to the formation of hundreds of polyps by the age of 30-40 years. Patients inevitably develop carcinoma. It is due to a mutation in a tumour suppressor gene called adenomatous polyposis coli gene (APC), located on chromosome 5. Genetic testing can be done by analysing DNA from a patient's white blood cells. Patients generally have a total colectomy with ileo-anal pouch formation in their twenties.

Patients with FAP are also at risk from duodenal tumours. A variant of FAP called Gardner's syndrome can also feature osteomas of the skull and mandible, retinal pigmentation, thyroid carcinoma and epidermoid cysts on the skin

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Peutz-Jeghers syndrome

Peutz-Jeghers syndrome is an autosomal dominant condition characterised by numerous hamartomatous polyps in the gastrointestinal tract. It is also associated with pigmented freckles on the lips, face, palms and soles. Around 50% of patients will have died from a gastrointestinal tract cancer by the age of 60 years.

Genetics

- autosomal dominant
- responsible gene encodes serine threonine kinase LKB1 or STK11

Features

- hamartomatous polyps in GI tract (mainly small bowel)
- pigmented lesions on lips, oral mucosa, face, palms and soles
- intestinal obstruction e.g. intussusception
- gastrointestinal bleeding

Management

- conservative unless complications develop

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[Peutz-Jeghers syndrome images](#)

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Distant Metastasis (M)

Questions

M0	No distant metastasis
M1	Distant metastasis
M1a	Metastasis confined to one organ or site (for example, liver, lung, ovary, nonregional node)
M1b	Metastases in more than one organ/site or the peritoneum

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Next question >

Colorectal cancer

Colorectal cancer is the third most common type of cancer in the UK and the second most cause of cancer deaths. Annually there are about 150,000 new cases diagnosed and 50,000 deaths from the disease.

Location of cancer (averages)

- rectal: 40%
- sigmoid: 30%
- descending colon: 5%
- transverse colon: 10%
- ascending colon and caecum: 15%

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Carcinoembryonic antigen may be used to monitor for recurrence in patients post-operatively or to assess response to treatment in patients with metastatic disease



Next question >

Colorectal cancer: screening

Overview

- most cancers develop from adenomatous polyps. Screening for colorectal cancer has been shown to reduce mortality by 16%
- the NHS now has a national screening programme offering screening every 2 years to all men and women aged 60 to 74 years in England, 50 to 74 years in Scotland. IPatients aged over 74 years may request screening
- eligible patients are sent faecal occult blood (FOB) tests through the post
- patients with abnormal results are offered a colonoscopy

At colonoscopy, approximately:

- 5 out of 10 patients will have a normal exam
- 4 out of 10 patients will be found to have polyps which may be removed due to their premalignant potential
- 1 out of 10 patients will be found to have cancer

Next question >





Angiodysplasia



Angiodysplasia is a vascular deformity of the gastrointestinal tract which predisposes to bleeding and iron deficiency anaemia. There is thought to be an association with aortic stenosis, although this is debated. Angiodysplasia is generally seen in elderly patients

Diagnosis

- colonoscopy
- mesenteric angiography if acutely bleeding

Management

- endoscopic cautery or argon plasma coagulation
- antifibrinolytics e.g. Tranexamic acid
- oestrogens may also be used

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Aortic stenosis, iron-deficiency anaemia - angiodysplasia

[Notes](#)

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A 35-year-old man presents with fatigue, erectile dysfunction and arthralgia. On examination he 'bronze' pigmentation of his skin and some early stigmata of chronic liver disease - haemochromatosis

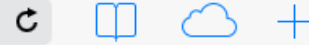
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Wilson's disease - chorea

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Question

Irritable bowel syndrome



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Increased

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Blocks

H2 antacids

Irreversible

84 factors

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Key factors

A 20-year-old

is relieved

syndrome

A woman

myasthenia

NICE published clinical guidelines on the diagnosis and management of irritable bowel syndrome (IBS) in 2008

The diagnosis of IBS should be considered if the patient has had the following for at least 6 months:

- abdominal pain, and/or
- bloating, and/or
- change in bowel habit

A positive diagnosis of IBS should be made if the patient has abdominal pain relieved by defecation or associated with altered bowel frequency stool form, in addition to 2 of the following 4 symptoms:

- altered stool passage (straining, urgency, incomplete evacuation)
- abdominal bloating (more common in women than men), distension, tension or hardness
- symptoms made worse by eating
- passage of mucus

Features such as lethargy, nausea, backache and bladder symptoms may also support the diagnosis

Red flag features should be enquired about:

- rectal bleeding
- unexplained/unintentional weight loss
- family history of bowel or ovarian cancer
- onset after 60 years of age

Suggested primary care investigations are:

- full blood count
- ESR/CRP
- coeliac disease screen (tissue transglutaminase antibodies)

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Irritable bowel syndrome: management

The management of irritable bowel syndrome (IBS) is often difficult and varies considerably between patients. NICE updated it's guidelines in 2015.

First-line pharmacological treatment - according to predominant symptom

- pain: antispasmodic agents
- constipation: laxatives but avoid lactulose
- diarrhoea: loperamide is first-line

For patients with constipation who are not responding to conventional laxatives linaclotide may be considered, if:

- optimal or maximum tolerated doses of previous laxatives from different classes have not helped and
- they have had constipation for at least 12 months

Second-line pharmacological treatment

- low-dose tricyclic antidepressants (e.g. amitriptyline 5-10 mg) are used in preference to selective serotonin reuptake inhibitors

Other management options

- psychological interventions - if symptoms do not respond to pharmacological treatments after 12 months and who develop a continuing symptom profile (refractory IBS), consider referring for cognitive behavioural therapy, hypnotherapy or psychological therapy
- complementary and alternative medicines: 'do not encourage use of acupuncture or reflexology for the treatment of IBS'

General dietary advice

- have regular meals and take time to eat
- avoid missing meals or leaving long gaps between eating
- drink at least 8 cups of fluid per day, especially water or other non-caffeinated drinks such as herbal teas
- restrict tea and coffee to 3 cups per day
- reduce intake of alcohol and fizzy drinks

Coeliac disease: investigation

Coeliac disease is caused by sensitivity to the protein gluten. Repeated exposure leads to villous atrophy which in turn causes malabsorption. Conditions associated with coeliac disease include dermatitis herpetiformis (a vesicular, pruritic skin eruption) and autoimmune disorders (type 1 diabetes mellitus and autoimmune hepatitis).

Diagnosis is made by a combination of immunology and jejunal biopsy. Villous atrophy and immunology normally reverses on a gluten-free diet.

NICE issued guidelines on the investigation of coeliac disease in 2009. If patients are already taking a gluten-free diet they should be asked, if possible, to reintroduce gluten for at least 6 weeks prior to testing.

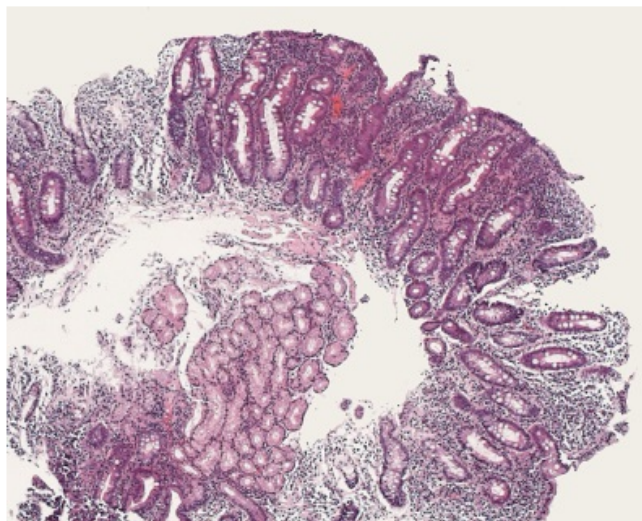
Immunology

- tissue transglutaminase (TTG) antibodies (IgA) are first-choice according to NICE
- endomysial antibody (IgA)
- anti-gliadin antibody (IgA or IgG) tests are not recommended by NICE
- anti-casein antibodies are also found in some patients

Jejunal biopsy

- villous atrophy
- crypt hyperplasia
- increase in intraepithelial lymphocytes
- lamina propria infiltration with lymphocytes

Rectal gluten challenge has been described but is not widely used



Next question >

Coeliac disease: management

The management of coeliac disease involves a **gluten-free diet**. Gluten containing cereals include:

- wheat: bread, pasta, pastry
- barley*: beer
- rye
- oats**

Some notable foods which are gluten-free include:

- rice
- potatoes
- corn (maize)

Tissue transglutaminase antibodies may be checked to check compliance with a gluten free diet.

Patients with coeliac disease often have a degree of **functional hyposplenism**. For this reason all patients with coeliac disease are offered the pneumococcal vaccine. Current guidelines suggest giving the influenza vaccine on an individual basis.

*whisky is made using malted barley. Proteins such as gluten are however removed during the distillation process making it safe to drink for patients with coeliac disease

**some patients with coeliac disease appear able to tolerate oats

Next question >

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IBD: histology

This histological differences between ulcerative colitis and Crohn's are summarised below:

Ulcerative colitis

- inflammation in mucosa and submucosa only (unless fulminant disease)
- widespread ulceration with preservation of adjacent mucosa which has the appearance of polyps ('pseudopolyps')
- inflammatory cell infiltrate in lamina propria
- crypt abscesses
- depletion of goblet cells and mucin from gland epithelium
- granulomas are infrequent

Crohn's

- inflammation occurs in all layers, down to the serosa. This predisposes to strictures, fistulas and adhesions
- oedema of mucosa and submucosa, combined with deep fissured ulcers ('rose-thorn') leads to a 'cobblestone' pattern
- lymphoid aggregates
- non-caseating granulomas

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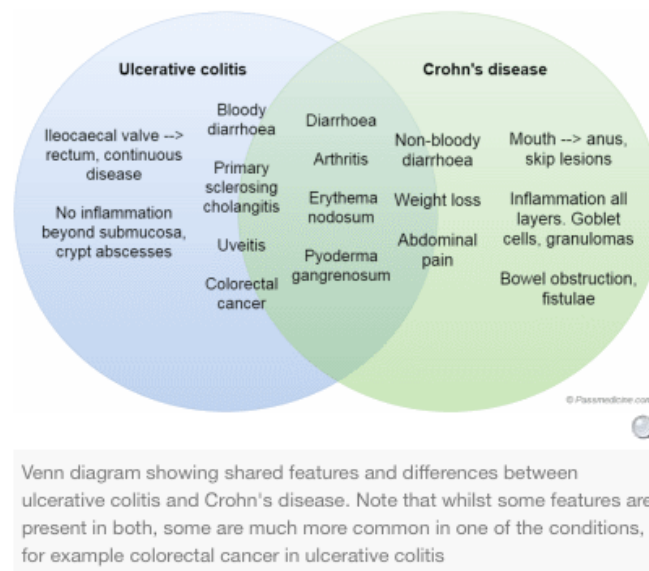
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Inflammatory bowel disease: key differences

The two main types of inflammatory bowel disease are Crohn's disease and Ulcerative colitis. They have many similarities in terms of presenting symptoms, investigation findings and management options.



There are however some key differences which are highlighted in table below:

	Crohn's disease (CD)	Ulcerative colitis (UC)
Features	Diarrhoea usually non-bloody Weight loss more prominent Upper gastrointestinal symptoms, mouth ulcers, perianal disease Abdominal mass palpable in the right iliac fossa	Bloody diarrhoea more common Abdominal pain in the left lower quadrant Tenesmus
Extra-intestinal	Gallstones are more common secondary to reduced bile acid reabsorption Oxalate renal stones*	Primary sclerosing cholangitis more common
Complications	Obstruction, fistula, colorectal cancer	Risk of colorectal cancer high in UC than CD

Extra-intestinal	Gallstones are more common secondary to reduced bile acid reabsorption Oxalate renal stones*	Primary sclerosing cholangitis more common
Complications	Obstruction, fistula, colorectal cancer	Risk of colorectal cancer high in UC than CD
Pathology	Lesions may be seen anywhere from the mouth to anus Skip lesions may be present	Inflammation always starts at rectum and never spreads beyond ileocaecal valve Continuous disease
Histology	Inflammation in all layers from mucosa to serosa - increased goblet cells - granulomas	No inflammation beyond submucosa (unless fulminant disease) - inflammatory cell infiltrate in lamina propria - neutrophils migrate through the walls of glands to form crypt abscesses - depletion of goblet cells and mucin from gland epithelium - granulomas are infrequent
Endoscopy	Deep ulcers, skip lesions - 'cobblestone' appearance	Widespread ulceration with preservation of adjacent mucosa which has the appearance of polyps ('pseudopolyps')
Radiology	Small bowel enema - high sensitivity and specificity for examination of the terminal ileum - strictures: 'Kantor's string sign' - proximal bowel dilation 'rose thorn' ulcers - fistulae	Barium enema - loss of haustrations - superficial ulceration, 'pseudopolyps' - long standing disease: colon is narrow and short - 'drainpipe colon'

*impaired bile acid reabsorption increases the loss calcium in the bile. Calcium normally binds oxalate.

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Ulcerative colitis

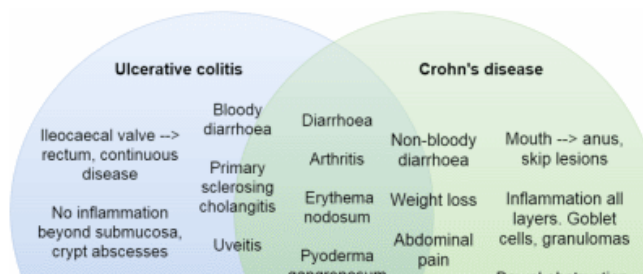
Ulcerative colitis (UC) is a form of inflammatory bowel disease. Inflammation always starts at rectum (hence it is the most common site for UC), never spreads beyond ileocaecal valve and is continuous. The peak incidence of ulcerative colitis is in people aged 15-25 years and in those aged 55-65 years.

The initial presentation is usually following insidious and intermittent symptoms. Features include:

- bloody diarrhoea
- urgency
- tenesmus
- abdominal pain, particularly in the left lower quadrant
- extra-intestinal features (see below)

Questions regarding the 'extra-intestinal' features of inflammatory bowel disease are common:

	Common to both Crohn's disease (CD) and Ulcerative colitis (UC)	Notes
Related to disease activity	Arthritis: pauciarticular, asymmetric Erythema nodosum Episcleritis Osteoporosis	Arthritis is the most common extra-intestinal feature in both CD and UC Episcleritis is more common in CD
Unrelated to disease activity	Arthritis: polyarticular, symmetric Uveitis Pyoderma gangrenosum Clubbing Primary sclerosing cholangitis	Primary sclerosing cholangitis is much more common in UC Uveitis is more common in UC



Ulcerative colitis: colorectal cancer

Overview

- risk of colorectal cancer is significantly higher than that of the general population although studies report widely varying rates
- the increased risk is mainly related to chronic inflammation
- worse prognosis than patients without ulcerative colitis (partly due to delayed diagnosis)
- lesions may be multifocal

Factors increasing risk of cancer

- disease duration > 10 years
- patients with pancolitis
- onset before 15 years old
- unremitting disease
- poor compliance to treatment

Colonoscopy surveillance in inflammatory bowel disease patients should be decided following risk stratification.

Lower risk

5 year follow up colonoscopy

- Extensive colitis with no active endoscopic/histological inflammation
- OR left sided colitis
- OR Crohn's colitis of <50% colon

Intermediate risk

3 year colonoscopy

- Extensive colitis with mild active endoscopy/histological inflammation
- OR post-inflammatory polyps
- OR family history of colorectal cancer in a first degree relative aged 50 or over

Higher risk

- disease duration > 10 years
- patients with pancolitis
- onset before 15 years old
- unremitting disease
- poor compliance to treatment

Colonoscopy surveillance in inflammatory bowel disease patients should be decided following risk stratification.

Lower risk

5 year follow up colonoscopy

- Extensive colitis with no active endoscopic/histological inflammation
- OR left sided colitis
- OR Crohn's colitis of <50% colon

Intermediate risk

3 year colonoscopy

- Extensive colitis with mild active endoscopy/histological inflammation
- OR post-inflammatory polyps
- OR family history of colorectal cancer in a first degree relative aged 50 or over

Higher risk

1 year follow up colonoscopy

- Extensive colitis with moderate/severe active endoscopic/histological inflammation
- OR stricture in past 5 years
- OR dysplasia in past 5 years declining surgery
- OR primary sclerosing cholangitis / transplant for primary sclerosing cholangitis
- OR family history of colorectal cancer in first degree relatives aged <50 years

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Ulcerative colitis: management

Treatment can be divided into inducing and maintaining remission. NICE released guidelines on the management of ulcerative colitis in 2013.

The severity of UC is usually classified as being mild, moderate or severe:

- mild: < 4 stools/day, only a small amount of blood
- moderate: 4-6 stools/day, varying amounts of blood, no systemic upset
- severe: >6 bloody stools per day + features of systemic upset (pyrexia, tachycardia, anaemia, raised inflammatory markers)

Inducing remission

- treatment depends on the extent and severity of disease
- rectal (topical) aminosaliclates or steroids: for distal colitis rectal mesalazine has been shown to be superior to rectal steroids and oral aminosaliclates
- oral aminosaliclates
- oral prednisolone is usually used second-line for patients who fail to respond to aminosaliclates. NICE recommend waiting around 4 weeks before deciding if first-line treatment has failed
- severe colitis should be treated in hospital. Intravenous steroids are usually given first-line

Maintaining remission

- oral aminosaliclates e.g. mesalazine
- azathioprine and mercaptopurine
- methotrexate is not recommended for the management of UC (in contrast to Crohn's disease)
- there is some evidence that probiotics may prevent relapse in patients with mild to moderate disease

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Crohn's disease: management

Crohn's disease is a form of inflammatory bowel disease. It commonly affects the terminal ileum and colon but may be seen anywhere from the mouth to anus. NICE published guidelines on the management of Crohn's disease in 2012.

General points

- patients should be strongly advised to stop smoking
- some studies suggest an increased risk of relapse secondary to NSAIDs and the combined oral contraceptive pill but the evidence is patchy

Inducing remission

- glucocorticoids (oral, topical or intravenous) are generally used to induce remission. Budesonide is an alternative in a subgroup of patients
- enteral feeding with an elemental diet may be used in addition to or instead of other measures to induce remission, particularly if there is concern regarding the side-effects of steroids (for example in young children)
- 5-ASA drugs (e.g. mesalazine) are used second-line to glucocorticoids but are not as effective
- azathioprine or mercaptopurine* may be used as an add-on medication to induce remission but is not used as monotherapy. Methotrexate is an alternative to azathioprine
- infliximab is useful in refractory disease and fistulating Crohn's. Patients typically continue on azathioprine or methotrexate
- metronidazole is often used for isolated peri-anal disease

Maintaining remission

- as above, stopping smoking is a priority (remember: smoking makes Crohn's worse, but may help ulcerative colitis)
- azathioprine or mercaptopurine is used first-line to maintain remission
- methotrexate is used second-line
- 5-ASA drugs (e.g. mesalazine) should be considered if a patient has had previous surgery

Surgery

- around 80% of patients with Crohn's disease will eventually have surgery
- see below for further detail

Surgery

- around 80% of patients with Crohn's disease will eventually have surgery
- see below for further detail

Surgical interventions in Crohn's disease

The commonest disease pattern in Crohn's is stricturing terminal ileal disease and this often culminates in an ileocaecal resection. Other procedures performed include segmental small bowel resections and stricturoplasty. Colonic involvement in patients with Crohn's is not common and, where found, distribution is often segmental. However, despite this distribution segmental resections of the colon in patients with Crohn's disease are generally not advocated because the recurrence rate in the remaining colon is extremely high, as a result, the standard options of colonic surgery in Crohn's patients are generally; sub total colectomy, panproctocolectomy and staged sub total colectomy and proctectomy. Restorative procedures such as ileoanal pouch have no role in therapy.

Crohn's disease is notorious for the development of intestinal fistulae; these may form between the rectum and skin (perianal) or the small bowel and skin. Fistulation between loops of bowel may also occur and result in bacterial overgrowth and malabsorption. Management of enterocutaneous fistulae involves controlling sepsis, optimising nutrition, imaging the disease and planning definitive surgical management.

Complications of Crohn's disease

As well as the well-documented complications described above, patients are also at risk of:

- small bowel cancer (standard incidence ratio = 40)
- colorectal cancer (standard incidence ratio = 2, i.e. less than the risk associated with ulcerative colitis)
- osteoporosis

*assess thiopurine methyltransferase (TPMT) activity before offering azathioprine or mercaptopurine

Next question >

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Crohn's disease

Crohn's disease is a form of inflammatory bowel disease. It commonly affects the terminal ileum and colon but may be seen anywhere from the mouth to anus.

Pathology

- cause is unknown but there is a strong genetic susceptibility
- inflammation occurs in all layers, down to the serosa. This is why patients with Crohn's are prone to strictures, fistulas and adhesions

Crohn's disease typically presents in late adolescence or early adulthood. Features include:

- presentation may be non-specific symptoms such as weight loss and lethargy
- diarrhoea: the most prominent symptom in adults. Crohn's colitis may cause bloody diarrhoea
- abdominal pain: the most prominent symptom in children
- perianal disease: e.g. Skin tags or ulcers
- extra-intestinal features are more common in patients with colitis or perianal disease

Investigations

- raised inflammatory markers
- increased faecal calprotectin
- anaemia
- low vitamin B12 and vitamin D

Questions regarding the 'extra-intestinal' features of inflammatory bowel disease are common:

	Common to both Crohn's disease (CD) and Ulcerative colitis (UC)	Notes
Related to disease activity	Arthritis: pauciarticular, asymmetric Erythema nodosum	Arthritis is the most common extra-intestinal feature in both CD and UC Episcleritis is more common in CD

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Crohn's disease: investigation

Crohn's disease is a form of inflammatory bowel disease. It commonly affects the terminal ileum and colon but may be seen anywhere from the mouth to anus

Bloods

- C-reactive protein correlates well with disease activity

Endoscopy

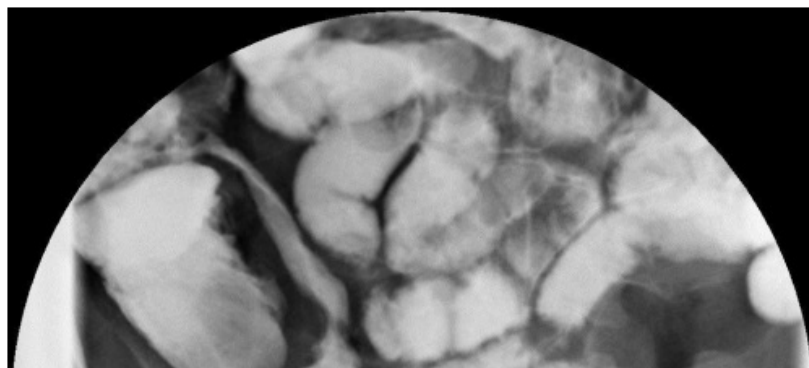
- colonoscopy is the investigation of choice
- features suggest of Crohn's include deep ulcers, skip lesions

Histology

- inflammation in all layers from mucosa to serosa
- goblet cells
- granulomas

Small bowel enema

- high sensitivity and specificity for examination of the terminal ileum
- strictures: 'Kantor's string sign'
- proximal bowel dilation
- 'rose thorn' ulcers
- fistulae



Crohn's disease: management

Crohn's disease is a form of inflammatory bowel disease. It commonly affects the terminal ileum and colon but may be seen anywhere from the mouth to anus. NICE published guidelines on the management of Crohn's disease in 2012.

General points

- patients should be strongly advised to stop smoking
- some studies suggest an increased risk of relapse secondary to NSAIDs and the combined oral contraceptive pill but the evidence is patchy

Inducing remission

- glucocorticoids (oral, topical or intravenous) are generally used to induce remission. Budesonide is an alternative in a subgroup of patients
- enteral feeding with an elemental diet may be used in addition to or instead of other measures to induce remission, particularly if there is concern regarding the side-effects of steroids (for example in young children)
- 5-ASA drugs (e.g. mesalazine) are used second-line to glucocorticoids but are not as effective
- azathioprine or mercaptopurine* may be used as an add-on medication to induce remission but is not used as monotherapy. Methotrexate is an alternative to azathioprine
- infliximab is useful in refractory disease and fistulating Crohn's. Patients typically continue on azathioprine or methotrexate
- metronidazole is often used for isolated peri-anal disease

Maintaining remission

- as above, stopping smoking is a priority (remember: smoking makes Crohn's worse, but may help ulcerative colitis)
- azathioprine or mercaptopurine is used first-line to maintain remission
- methotrexate is used second-line
- 5-ASA drugs (e.g. mesalazine) should be considered if a patient has had previous surgery

Surgery

- around 80% of patients with Crohn's disease will eventually have surgery
- see below for further detail

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Surgery

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- see below for further detail

Surgical interventions in Crohn's disease

The commonest disease pattern in Crohn's is stricturing terminal ileal disease and this often culminates in an ileocaecal resection. Other procedures performed include segmental small bowel resections and stricturoplasty. Colonic involvement in patients with Crohn's is not common and, where found, distribution is often segmental. However, despite this distribution segmental resections of the colon in patients with Crohn's disease are generally not advocated because the recurrence rate in the remaining colon is extremely high, as a result, the standard options of colonic surgery in Crohn's patients are generally; sub total colectomy, panproctocolectomy and staged sub total colectomy and proctectomy. Restorative procedures such as ileoanal pouch have no role in therapy.

Crohn's disease is notorious for the development of intestinal fistulae; these may form between the rectum and skin (perianal) or the small bowel and skin. Fistulation between loops of bowel may also occur and result in bacterial overgrowth and malabsorption. Management of enterocutaneous fistulae involves controlling sepsis, optimising nutrition, imaging the disease and planning definitive surgical management.

Complications of Crohn's disease

As well as the well-documented complications described above, patients are also at risk of:

- small bowel cancer (standard incidence ratio = 40)
- colorectal cancer (standard incidence ratio = 2, i.e. less than the risk associated with ulcerative colitis)
- osteoporosis

*assess thiopurine methyltransferase (TPMT) activity before offering azathioprine or mercaptopurine

Next question >

Factors increasing risk of cancer

- disease duration > 10 years
- patients with pancolitis
- onset before 15 years old
- unremitting disease
- poor compliance to treatment

Colonoscopy surveillance in inflammatory bowel disease patients should be decided following risk stratification.

Lower risk

5 year follow up colonoscopy

- Extensive colitis with no active endoscopic/histological inflammation
- OR left sided colitis
- OR Crohn's colitis of <50% colon

Intermediate risk

3 year colonoscopy

- Extensive colitis with mild active endoscopy/histological inflammation
- OR post-inflammatory polyps
- OR family history of colorectal cancer in a first degree relative aged 50 or over

Higher risk

1 year follow up colonoscopy

- Extensive colitis with moderate/severe active endoscopic/histological inflammation
- OR stricture in past 5 years
- OR dysplasia in past 5 years declining surgery
- OR primary sclerosing cholangitis / transplant for primary sclerosing cholangitis
- OR family history of colorectal cancer in first degree relatives aged <50 years

Next question >

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Quick comparison: Drug adverse effects
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| Parkinsonism <ul style="list-style-type: none"> • metoclopramide • antipsychotics | Disulfiram-like reaction with alcohol <ul style="list-style-type: none"> • metronidazole |
|--|--|

Relevant to my revision

Less relevant

Metoclopramide

Metoclopramide is a D2 receptor antagonist mainly used in the management of nausea. Other uses include:

- gastro-oesophageal reflux disease
- prokinetic action is useful in gastroparesis secondary to diabetic neuropathy
- often combined with analgesics for the treatment of migraine (migraine attacks result in gastroparesis, slowing the absorption of analgesics)

Adverse effects

- extrapyramidal effects: oculogyric crisis. This is particularly a problem in children and young adults
- hyperprolactinaemia
- tardive dyskinesia
- parkinsonism

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Cholestyramine



Cholestyramine is a bile acid sequestrant used in the management of hyperlipidaemia. It decreases bile acid reabsorption in the small intestine, therefore upregulating the amount of cholesterol that is converted to bile acid. The main effect it has on the lipid profile is to reduce LDL cholesterol. It is also occasionally used in Crohn's disease for treatment resistant diarrhoea.

Adverse effects

- abdominal cramps and constipation
- decreases absorption of fat-soluble vitamins
- cholesterol gallstones
- may raise level of triglycerides

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Key facts from this session are shown below (score/attempts), click on the fact for background information

Cholestyramine may cause constipation

[Notes](#)

(1/1)

Liver cysts - *Echinococcus granulosus* infection[Notes](#)

(1/1)

Metoclopramide may cause parkinsonism

[Notes](#)

(1/1)

Abdominal pain

The table below gives characteristic exam question features for conditions causing abdominal pain. Unusual and 'medical' causes of abdominal pain should also be remembered:

- myocardial infarction
- diabetic ketoacidosis
- pneumonia
- acute intermittent porphyria
- lead poisoning

Condition	Characteristic exam feature
Peptic ulcer disease	Duodenal ulcers: more common than gastric ulcers, epigastric pain relieved by eating Gastric ulcers: epigastric pain worsened by eating Features of upper gastrointestinal haemorrhage may be seen (haematemesis, melena etc)
Appendicitis	Pain initial in the central abdomen before localising to the right iliac fossa Anorexia is common Tachycardia, low-grade pyrexia, tenderness in RIF Rovsing's sign: more pain in RIF than LIF when palpating LIF
Acute pancreatitis	Usually due to alcohol or gallstones Severe epigastric pain Vomiting is common Examination may reveal tenderness, ileus and low-grade fever Periumbilical discolouration (Cullen's sign) and flank discolouration (Grey-Turner's sign) is described but rare
Biliary colic	Pain in the RUQ radiating to the back and interscapular region, may be following a fatty meal. Slight misnomer as the pain may persist for hours Obstructive jaundice may cause pale stools and dark urine It is sometimes taught that patients are female, forties, fat and fair although this is obviously a generalisation
Acute cholecystitis	History of gallstones symptoms (see above) Continuous RUQ pain Fever, raised inflammatory markers and white cells Murphy's sign positive (arrest of inspiration on palpation of the RUQ)
Diverticulitis	Colicky pain typically in the LLQ Fever, raised inflammatory markers and white cells
Abdominal aortic aneurysm	Severe central abdominal pain radiating to the back Presentation may be catastrophic (e.g. Sudden collapse) or sub-acute (persistent severe central abdominal pain with developing shock)

	Obstructive jaundice may cause pale stools and dark urine It is sometimes taught that patients are female, forties, fat and fair although this is obviously a generalisation
Acute cholecystitis	History of gallstones symptoms (see above) Continuous RUQ pain Fever, raised inflammatory markers and white cells Murphy's sign positive (arrest of inspiration on palpation of the RUQ)
Diverticulitis	Colicky pain typically in the LLQ Fever, raised inflammatory markers and white cells
Abdominal aortic aneurysm	Severe central abdominal pain radiating to the back Presentation may be catastrophic (e.g. Sudden collapse) or sub-acute (persistent severe central abdominal pain with developing shock) Patients may have a history of cardiovascular disease
Intestinal obstruction	History of malignancy/previous operations Vomiting Not opened bowels recently 'Tinkling' bowel sounds

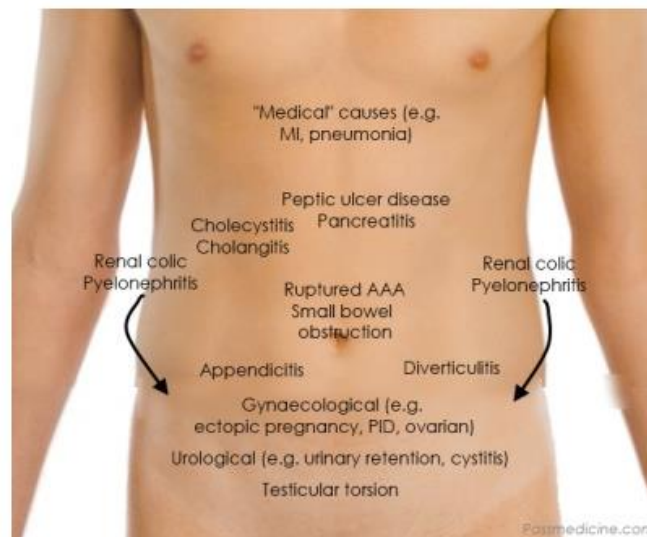


Diagram showing stereotypical areas where particular conditions present. The diagram is not exhaustive and only lists the more common conditions seen in clinical practice. Note how pain from renal causes such as renal/ureteric colic and pyelonephritis may radiate and move from the loins towards the suprapubic area.

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Aminosalicylate drugs

5-aminosalicylic acid (5-ASA) is released in the colon and is not absorbed. It acts locally as an anti-inflammatory. The mechanism of action is not fully understood but 5-ASA may inhibit prostaglandin synthesis

Sulphasalazine

- a combination of sulphapyridine (a sulphonamide) and 5-ASA
- many side-effects are due to the sulphapyridine moiety: rashes, oligospermia, headache, Heinz body anaemia, megaloblastic anaemia, lung fibrosis
- other side-effects are common to 5-ASA drugs (see mesalazine)

Mesalazine

- a delayed release form of 5-ASA
- sulphapyridine side-effects seen in patients taking sulphasalazine are avoided
- mesalazine is still however associated with side-effects such as GI upset, headache, agranulocytosis, pancreatitis*, interstitial nephritis

Olsalazine

- two molecules of 5-ASA linked by a diazo bond, which is broken by colonic bacteria

*pancreatitis is 7 times more common in patients taking mesalazine than sulfasalazine

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[Adverse effects of sulfasalazine and mesalazine](#)

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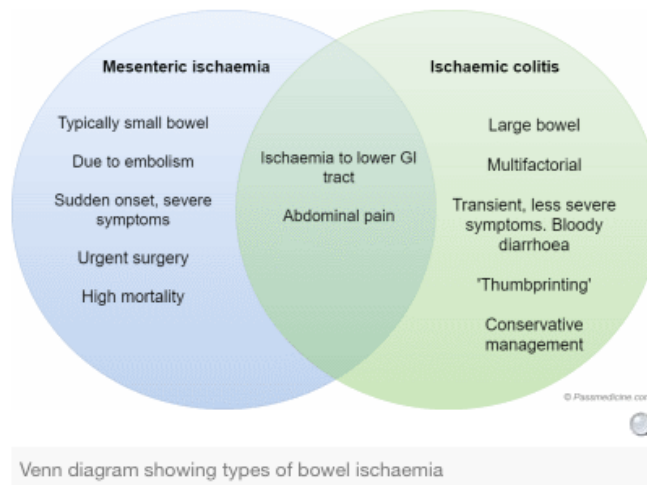
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Ischaemia to the lower gastrointestinal tract

Ischaemia to the lower gastrointestinal tract can result in a variety of clinical conditions. Whilst there is no standard classification it can be useful to separate cases into 3 main conditions

- acute mesenteric ischaemia
- chronic mesenteric ischaemia
- ischaemic colitis



Common features in bowel ischaemia

Common predisposing factors

- increasing age
- atrial fibrillation - particularly for mesenteric ischaemia
- other causes of emboli: endocarditis, malignancy
- cardiovascular disease risk factors: smoking, hypertension, diabetes
- cocaine: ischaemic colitis is sometimes seen in young patients following cocaine use

Common features

- abdominal pain - in acute mesenteric ischaemia this is often of sudden onset, severe and out-of-keeping with physical exam findings
- rectal bleeding
- diarrhoea
- fever
- bloods typically show an elevated white blood cell count associated with a lactic acidosis

Diagnosis

- CT is the investigation of choice

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Carcinoid tumours

Carcinoid syndrome

- usually occurs when metastases are present in the liver and release serotonin into the systemic circulation
- may also occur with lung carcinoid as mediators are not 'cleared' by the liver

Features

- flushing (often earliest symptom)
- diarrhoea
- bronchospasm
- hypotension
- right heart valvular stenosis (left heart can be affected in bronchial carcinoid)
- other molecules such as ACTH and GHRH may also be secreted resulting in, for example, Cushing's syndrome
- pellagra can rarely develop as dietary tryptophan is diverted to serotonin by the tumour

Investigation

- urinary 5-HIAA
- plasma chromogranin A y

Management

- somatostatin analogues e.g. octreotide
- diarrhoea: cyproheptadine may help

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Clostridium difficile

Clostridium difficile is a Gram positive rod often encountered in hospital practice. It produces an exotoxin which causes intestinal damage leading to a syndrome called pseudomembranous colitis. *Clostridium difficile* develops when the normal gut flora are suppressed by broad-spectrum antibiotics. Clindamycin is historically associated with causing *Clostridium difficile* but the aetiology has evolved significantly over the past 10 years. Second and third generation cephalosporins are now the leading cause of *Clostridium difficile*.

Features

- diarrhoea
- abdominal pain
- a **raised white blood cell count** is characteristic
- if severe toxic megacolon may develop

Diagnosis is made by detecting *Clostridium difficile* toxin (CDT) in the stool

Management

- first-line therapy is oral metronidazole for 10-14 days
- if severe or not responding to metronidazole then oral vancomycin may be used
- for life-threatening infections a combination of oral vancomycin and intravenous metronidazole should be used



Next question >

Skin disorders associated with malignancy

Paraneoplastic syndromes associated with internal malignancies:

Skin disorder	Associated malignancies
Acanthosis nigricans	Gastric cancer
Acquired ichthyosis	Lymphoma
Acquired hypertrichosis lanuginosa	Gastrointestinal and lung cancer
Dermatomyositis	Ovarian and lung cancer
Erythema gyratum repens	Lung cancer
Erythroderma	Lymphoma
Migratory thrombophlebitis	Pancreatic cancer
Necrolytic migratory erythema	Glucagonoma
Pyoderma gangrenosum (bullous and non-bullous forms)	Myeloproliferative disorders
Sweet's syndrome	Haematological malignancy e.g. Myelodysplasia - tender, purple plaques
Tylosis	Oesophageal cancer

Next question >

Acute mesenteric ischaemia

Acute mesenteric ischaemia is typically caused by an embolism resulting in occlusion of an artery which supplies the small bowel, for example the superior mesenteric artery. Classically patients have a history of atrial fibrillation.

The abdominal pain is typically severe, of sudden onset and out-of-keeping with physical exam findings.

Management

- urgent surgery is usually required
- poor prognosis, especially if surgery delayed

Chronic mesenteric ischaemia

Chronic mesenteric ischaemia is a relatively rare clinical diagnosis due to its non-specific features and may be thought of as 'intestinal angina'. Colicky, intermittent abdominal pain occurs.

Ischaemic colitis

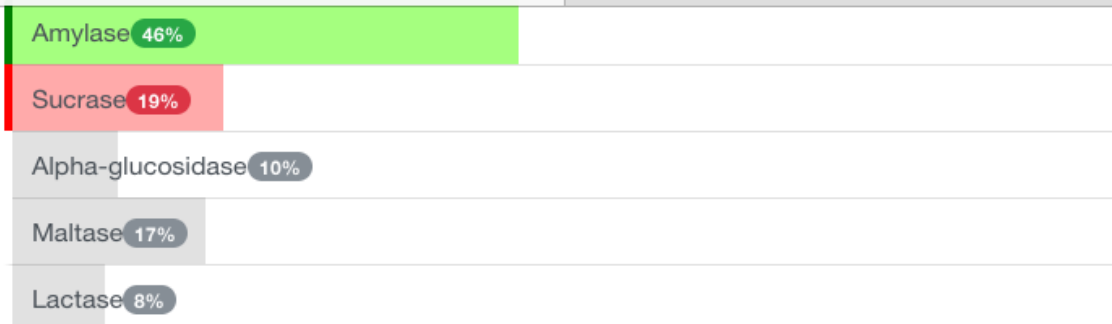
Ischaemic colitis describes an acute but transient compromise in the blood flow to the large bowel. This may lead to inflammation, ulceration and haemorrhage. It is more likely to occur in 'watershed' areas such as the splenic flexure that are located at the borders of the territory supplied by the superior and inferior mesenteric arteries.

Investigations

- 'thumbprinting' may be seen on abdominal x-ray due to mucosal oedema/haemorrhage

Management

- usually supportive
- surgery may be required in a minority of cases if conservative measures fail. Indications would include generalised peritonitis, perforation or ongoing haemorrhage



Amylase: breaks starch down to sugars



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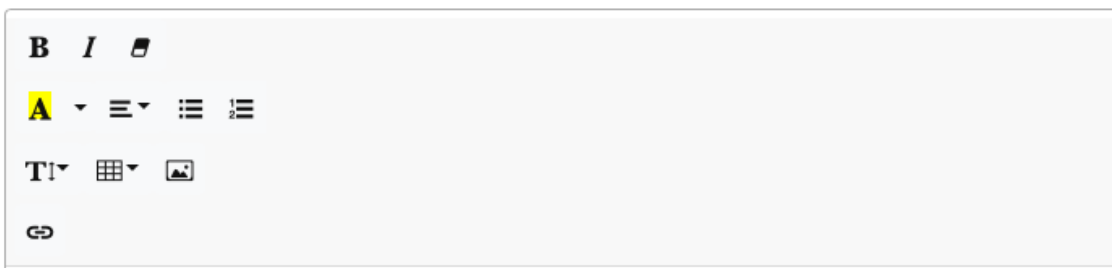
Gastrointestinal physiology: enzymes

Amylase is present in saliva and pancreatic secretions. It breaks starch down into sugar

The following brush border enzymes are involved in the breakdown of carbohydrates:

- maltase: cleaves disaccharide maltose to glucose + glucose
- sucrase: cleaves sucrose to fructose and glucose
- lactase: cleaves disaccharide lactose to glucose + galactose

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